ASSOCIATIONS BETWEEN ANXIETY AND NEUROPHYSIOLOGICAL MEASURES OF COGNITIVE CONTROL ACROSS DEVELOPMENT

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ABSTRACT

Cognitive control, or the ability to monitor performance and recruit and maintain cognitive processes to complete tasks, is theorized to relate to anxiety symptom development in youth. Anxiety has also been proposed to impact cognitive control in youth. Notably, the majority of electroencephalogram (EEG) research that has examined the association between anxiety and cognitive control has focused on a single event-related potential, the error-related negativity (ERN). Research examining the association between ERN and anxiety in youth has been largely mixed, and, as a result, age has been proposed as a moderator of this association in youth. However, age moderation has seldom been tested and the majority of research examining anxiety and the ERN in youth has been cross sectional in nature. Time-frequency (TF) analysis of EEG data has emerged as a novel method to examine timing and strength of neural oscillations relevant to cognitive control and anxiety. The aim of the current dissertation was two-fold: 1) to examine associations between anxiety and multiple measures of cognitive control, including the ERN, several TF metrics, and task behavior, and 2) to test age moderation of these associations. I analyzed data from a longitudinal study of 168 community youth ages 3 – 13 years old that completed a developmentally- appropriate Go/No Go Task at baseline, 18-month follow-up and 36-month follow-up. Generalized anxiety disorder and social anxiety symptom symptoms were the focus of my analysis, because these symptoms have previously been shown to relate to the ERN in youth. Contrary to hypotheses, anxiety did not relate to measures of cognitive control at baseline or longitudinally, and this association was not moderated by baseline age or aging (*p*s> 0.05). I suggest that the associations between anxiety and cognitive control may be nuanced and point to directions for future investigation, including exploring stressors, development, and sex as moderators, as well as considering diverse measures of cognitive control and anxiety symptom

severity. Understanding how and for whom anxiety relates to cognitive control will ultimately lead to more tailored and targeted clinical applications.

This dissertation is dedicated to my husband, my sister and my parents.
Thank you all for offering a listening ear and support during times of challenge and always reminding me of the bigger picture that inspires me and carries me forward.

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TABLE OF CONTENTS

Introduction

The impact of anxiety disorders in youth has been the subject of growing concern. The US Preventative Service Task Force recently recommended that all youth ages eight and older be screened for anxiety disorders to increase likelihood of early intervention and better long-term mental health outcomes (US Preventive Services Task Force, 2022). Indeed, five to 11% percent of youth are estimated to have an anxiety disorder and anxiety disorders with onset in youth often persist into adulthood (American Psychiatric Association, 2013; Baxter et al., 2013; Beesdo et al., 2009). Commonly-cited risk factors for anxiety disorders include genetic predisposition, caregiving/parenting-related factors, and experiences of trauma. While these are certainly significant factors, an often-neglected factor associated with anxiety development is self-regulation. *Self-regulation* is the ability to modulate one's own internal states and behavior to regulate reactivity. It has been theorized that reduced self-regulation relates to greater anxiety symptoms among individuals with anxious temperament (i.e., high neuroticism; e.g., Muris & Ollendick, 2005). Reduced ability to self-regulate has also been theorized to occur as a consequence of anxiety (Eysenck et al., 2007). Specifically, anxiety is thought to usurp resources otherwise used for cognitive processes (Eysenck et al., 2007), including those that support engagement in self-regulation. Regardless of directionality, better understanding this association offers an important avenue for clarifying anxiety development and its impacts in youth.

Empirical evidence from behavioral and self/informant-report studies indicates better ability to self-regulate relates to fewer anxiety symptoms in children and adolescents (e.g., Muris, 2006; Visu-Petra et al., 2006). Research examining this association from a neurophysiological perspective has suggested the association may be more nuanced, such that the ability to self-regulate in the context of anxiety may develop as neural systems that support self-regulation

become more efficient (Moser, 2017). However, neurophysiological investigation has often been limited to a single neural signal (i.e., the error-related negativity), seldom employed a longitudinal approach, and rarely considered the role of development. More thorough neurophysiological research is needed to gain critical insights into the mechanisms of anxiety disorder development in youth. In the current dissertation, I utilize a multimodal approach incorporating behavioral and multiple neurophysiological measures of self-regulation to better understand how and for whom anxiety and self-regulation relate during development.

Self-Regulation: Effortful Control & Cognitive Control

Effortful Control (EC; Rueda, 2012) is a self-regulation component of temperament that emerges after the first year of life and continues to develop throughout childhood. EC is thought to be comprised of several abilities, including conflict resolution, error-monitoring, inhibitory control, voluntary focus, shifting of attention, and taking pleasure from low-intensity stimuli (Rothbart et al., 2007; Rueda et al., 2010).

While EC has long been studied within the temperament literature, it has been hypothesized to be closely related to cognitive control, a concept heavily researched in cognitive psychology and neuroscience (Nigg, 2017). *Cognitive control* (CC) is the ability to engage in functions that "encode and maintain a representation of the current task" and recruit other cognitive and perceptual processes necessary for the task at hand (Botvinick & Braver, 2015, p. 85). Shenhav et al. (2013) detail three primary functions of cognitive control. First, cognitive control consists of monitoring how well current processes are meeting task demands, from both external (e.g., task instructions) and internal (e.g., motivationally-relevant valuation of payoffs) sources (Shenhav et al., 2013). The instantiation of cognitive control processes is often prompted by detection of a conflict, which can occur in the response, perceptual, cognitive/internal, or goal-

related domains, that indicates a change is needed (Inzlicht et al., 2015; Nigg, 2017; Shenhav et al., 2013). Second, following detection that some change is needed, cognitive control engages in a decision process that selects the control signals necessary to complete the task(s) at hand (Shenhav et al., 2013). It is hypothesized to do so by taking into account potential payoff or the value of achieving the outcome with the cost of engaging in cognitive control at a particular intensity (Shenhav et al., 2013). The magnitude and direction of the signal selected represents that which maximizes the expected value of control (Shenhav et al., 2013). Finally, cognitive control exercises a regulatory function by influencing how information is processed by lower-level cognitive processes (e.g., attention; Shenhav et al., 2013). For example, this may include biasing attention or providing templates for memory searches (Shenhav et al., 2013). Notably, Stuss (1992) proposed a similar model using a developmental framework.

Although EC has typically been assessed via self-report or informant-report measures, scores from these questionnaires are associated with performance on behavioral tasks used to assess CC (Rothbart et al., 2007; Rueda, 2012; Rueda et al., 2010). Thus, EC and CC are thought to be strongly overlapping constructs, despite their origins in disparate areas of psychology.

Anxiety and Behavioral & Self-Report Measures of EC/CC

Importantly, self-reported individual differences in EC/CC have been associated with academic, behavioral and socioemotional outcomes, including psychopathology (Rueda et al., 2010). Greater capacity for EC/CC is generally considered to promote academic success, increase engagement in prosocial behaviors, and reduce psychopathology (Rueda et al., 2010). Therefore, individual differences in EC/CC are crucial to consider in the context of anxiety symptoms, both as a potential risk/protective factor for anxiety and as consequences of anxiety.

A wealth of literature has examined the association between anxiety symptoms and various aspects of EC/CC in youth. Anxiety relates to poorer CC as measured by performance on cognitive tasks in children and adolescents (for reviews: Songco, Hudson, & Fox, 2020; Visu-Petra, Ciairano, & Miclea, 2006). Moreover, the association between temperamental negative affect and anxiety symptoms is moderated by self- and parent-reported effortful control, such that anxiety symptoms emerge when negative affect is high and effortful control is low (Muris, 2006; Muris et al., 2007; for reviews: Lonigan & Phillips, 2001; Muris & Ollendick, 2005).

The Error-Related Negativity (ERN)

Studies have shown that anxiety symptoms relate to neural measures of EC/CC. A body of work in youth has shown an association between anxiety symptoms and the error-related negativity (ERN), a neural response measured using electroencephalogram (EEG) that occurs approximately 0 – 100ms at frontocentral sites after an error is made (for reviews: Meyer, 2017; Moser, 2017). Source localization studies have suggested that the ERN is primarily generated by the ACC (for review: Gehring et al., 2012; Lo, 2018), a region that has been theorized to play a primary role in CC (e.g., Botvinick & Braver, 2015; Shenhav, Botvinick, & Cohen, 2013).

There are several theories that describe the function of the ACC and the significance of the ERN. Early researchers of the ERN suggested that it reflects the process of comparing the erroneous response to the estimated correct response, such that the ACC is a comparator between the responses (i.e., the Error Detection/Comparator Theory; for review: Gehring, Liu, Orr, & Carp, 2012). Yeung et al. (2004), on the other hand, proposed the Conflict-Monitoring Theory, theorizing the ERN arises from the co-activation of the erroneous response and the subsequent corrective response after an error is made. The conflict generated by this co-activation of responses is detected by the ACC and signals the need for greater cognitive control on the next

trial (for review: Gehring, Liu, Orr, & Carp, 2012; Yeung, Botvinick, & Cohen, 2004). Holroyd and Coles proposed the Reinforcement Learning Theory of the ERN, which suggests that the ERN occurs as a result of an unexpected, negative event detected by the basal ganglia (2002). The basal ganglia then communicates that this negative event has occurred to the ACC via the midbrain dopaminergic system, resulting in the generation of the ERN (Holroyd et al., 2005; Holroyd & Coles, 2002). The ERN is therefore a signal that communicates the need to improve task performance (Holroyd et al., 2005; Holroyd & Coles, 2002). In contrast, the Predicted Response Outcome (PRO) model proposes that the ACC serves to predict the likelihood of certain events and elicits a signal in the absence of the expected outcome (i.e., in the case of a surprising outcome), such as an error (Brown, 2013). Finally, in the Expected Value of Control theory, Shenhav et al. (2013) proposed that the dACC plays a primary role in interrupting on-going default behavior and determining both the intensity and the nature of the control signals selected in a particular situation. The dACC may also be involved in the specification of the control signal used. Its activity differs dependent on state-relevant factors, such as task rules and specific actions, as well as has been suggested to be sensitive to the valuation of outcomes relevant to assess for the cost of control (for review: Shenhav et al., 2013). An extension of this theory suggests that the ERN is a signal that specifies the intensity and nature of cognitive control required in a given situation (Moser et al., 2013).

Irrespective of theory, the ERN is an ACC signal indicating that greater CC is needed after mistakes (Gehring et al., 2012). Consistent with this notion emerging from the adult literature, the ERN has been described by some as an index of EC development in children (for review: Lo, 2018). The ERN increases in amplitude with age and is reflective of increased neural efficiency (for review: Lo, 2018).

The Error-Related Negativity (ERN) and Anxiety

A bulk of research in adults has indicated that anxiety is related to a larger ERN amplitude (for reviews: Moser et al., 2013, 2016; Saunders & Inzlicht, 2020). Two theories offer frameworks for interpreting the ERN in the context of anxiety. The compensatory error-monitoring hypothesis (CEMH) asserts that a larger ERN amplitude in anxiety reflects a call for increased cognitive effort in order to compensate for the cognitive load of worrisome anxious thoughts (Moser et al., 2013). Worry is hypothesized to co-opt working memory resources that would otherwise be devoted to engaging in the task at hand (Eysenck et al., 2007; Moser et al., 2013). More resources are then recruited to perform the task adequately for anxious individuals (Eysenck et al., 2007; Moser et al., 2013). In contrast, the endogenous threat perspective asserts that the ERN is enhanced in anxiety because of increased sensitivity to endogenous threat (Weinberg et al., 2016). Errors are perceived as threatening to anxious individuals, resulting in an enhanced ERN (Weinberg et al., 2016). Differences in the interpretation of the ERN itself and the ERN in the context of anxiety have resulted in the ERN being considered under three separate domains of the Research Domain Criteria (RDoC): Cognitive Systems (Cognitive Control), Negative Affect Systems (Sustained Threat) and Positive Valence Systems (Reward Learning). Despite these differences, both theories of enlarged ERN in anxiety posit that an increased ERN signifies the need for greater CC engagement following errors in anxious individuals (Moser et al., 2013; Weinberg et al., 2016).

In youth, the association between anxiety and the ERN has been shown to differ by age (Meyer, 2017; Moser, 2017). In children older than age nine, symptoms of generalized anxiety disorder (GAD; e.g., worry) and social anxiety are correlated with an *enlarged* ERN amplitude (Hanna et al., 2020; for reviews: Meyer, 2017; Moser, 2017). The direction of this association in

older children mirrors that found in adults. In contrast, mixed findings have been found in youth under age 9. Two studies have found that greater anxiety/fear behaviors relate to a *smaller* ERN amplitude in children 5 – 8 years old (Lo et al., 2016; Moser et al., 2015; Torpey et al., 2013). One study that examined age as a moderator of the relationship found no association between anxiety and the ERN in younger children (approx. ages 8 – 9 years), but a significant association between anxiety and a larger ERN in older children (approx. ages 10-13 years; (Meyer et al., 2012). Yet another study found that children ages 6 years old with an anxiety disorder had a larger ERN than age-matched controls (Meyer et al., 2013).

It has been hypothesized that the reason anxiety becomes more related to an enlarged ERN amplitude as children age is because CC/EC becomes more developed and coordinated with motivational and affective systems (for review: Moser, 2017). Several lines of research support this notion. As previously reviewed, the ERN amplitude increases with age (DuPuis et al., 2015; Lo, 2018). Functional magnetic resonance imaging (fMRI) evidence suggests that dACC activity increases over childhood and early adolescence and that increased dACC activity is correlated with improvements in inhibition across development (Crone & Steinbeis, 2017; Luna et al., 2015). Research also indicates that areas responsible for conveying information about salience (i.e., insula) and valuation (i.e., orbitofrontal cortex and inferior frontal gyrus) to the ACC become more active with development (Braver, 2012; Moser, 2017; Shenhav et al., 2013). Thus, this increased input may play a role in the growth of the ERN across development (Moser 2017). Importantly, increased valuation and saliency information in older children may prompt increased CC engagement (i.e., a larger ERN) to overcome anxious thoughts (Moser, 2017). Younger children are, therefore, theorized to be unable to effectively engage in the same

compensatory effort in response to anxiety as older children and adults (for review: Moser, 2017).

Others have hypothesized, however, that an enhanced ERN with age reflects an increased ability to experience internally generated threats (such as errors) as salient and aversive (Meyer, 2017). Youth are speculated to experience a normative developmental shift from fear of external threat (e.g., fear of the dark) to fear of internal threat (e.g., worry; Meyer, 2017) between the ages of 8 – 9 years old. Because the ERN is characterized as a signal of endogenous/internal threat under this theory, anxiety is hypothesized to relate to a larger ERN in youth older than age 9.

Despite the promise of the ERN as a neurophysiological index of CC/EC processes involved in anxiety, it only reflects time-domain information occurring around the erroneous response (Cohen, 2014; Luck, 2014). The ERN and other event-related potential analyses assume that signals are phase-locked from trial-to-trial, such that the timing of the signal is assumed to be highly similar from trial to trial (Cohen, 2014; Luck, 2014). Time-frequency (TF) analyses, on the other hand, offer the ability to capture multiple aspects of cognitive function through examining additional aspects of neural oscillations.

Time Frequency (TF) Analyses

TF analyses extract distinct information from neural oscillations, including frequency, phase and power information. Frequency is the speed of the oscillations expressed in Hz, which is the number of cycles per second (Cohen, 2014). Phase is defined as the position along the sine wave at any given time point measured in radians or degrees (Cohen, 2014). Power is the amount of energy in the frequency band calculated through taking the squared amplitude of the oscillation (Cohen, 2014).

TF analyses allow for unique information to be extracted from EEG data by (1) being able to target activity within specific frequency bands which may hold particular relevance to CC and (2) distinguishing phase information from amplitude information in neural oscillations, allowing for the signal timing and the signal strength to be examined independently (Cohen, 2014; Morales et al., 2022; Watts et al., 2018). The majority of studies that utilize TF analyses of EEG to investigate CC have examined neural activity occurring within the theta band (4 – 8 Hz). Activity in the theta band has been hypothesized to be a critical mechanism for CC (Cavanagh & Frank, 2014; Morales et al., 2022). Thus, TF analyses examining activity in the theta band provide additional meaningful indices of CC processes.

Theta Power. Several metrics can be obtained using TF analyses to examine signal timing and strength in the theta band and provide further insights into CC. Power in the theta band at frontal midline sites provides an index of the strength of the neural signal (Cavanagh & Frank, 2014; Morales et al., 2022). Power can be measured in two distinct ways, both of which have been examined after errors are made in youth (Buzzell et al., 2019). Evoked/average power following errors primarily indexes phase-locked information (i.e., power from signals that occur with the same timing on each trial) within the theta band (Buzzell et al., 2019). In contrast, total power after errors measures the signal strength of both phase- and non-phase-locked information within the theta frequency band (Buzzell et al., 2019). Both evoked power and total power in the theta band are increased on errors compared to correct trials in youth on speeded two-choice tasks (Buzzell et al., 2019; DuPuis et al., 2015; Gavin et al., 2019; Morales et al., 2022). However, mixed findings have been identified with regards to the impact of development on theta power. A longitudinal study of Kindergarten through second-grade children showed that total power on error trials decreased with age (DuPuis et al., 2015). A cross-sectional study of 7

to 25 year olds also identified a decrease in evoked power on error-trials with age, but only after correcting for differences in signal latency (Gavin et al., 2019). In contrast, a recent large cross-sectional study of 4 – 9-year-old children showed that total power on errors increased with age (Morales et al., 2022). Differences in findings may be explained by differences in age ranges, tasks utilized, and TF analytic approaches.

Inter-trial Phase Synchrony (ITPS). Inter-trial phase synchrony (ITPS) is a TF measure of signal timing consistency from trial to trial (Cohen, 2014; Morales et al., 2022). Theta ITPS is assessed by examining the consistency of phase oscillations in the theta frequency band across trials (Cohen, 2014; Morales et al., 2022). Theta ITPS has been shown to be enhanced on error trials and to increase with age in two studies of youth (DuPuis et al., 2015; Gavin et al., 2019). Thus, greater theta ITPS may reflect greater coordination and efficiency of neural activity with age (DuPuis et al., 2015). Notably, one study identified null findings, such that consistency in signal timing did not vary with age on error and correct trials in young children (Morales et al., 2022). Again, inconsistencies in findings may be explained by differing methodological approaches.

Inter-channel Phase Synchrony (ICPS). Finally, theta inter-channel phase synchrony (ICPS) examines the consistency in phase oscillations between different recording sites (Cohen, 2014; Morales et al., 2022). ICPS is thought to reflect a mechanism for neural communication whereby functional brain networks become coordinated (Cavanagh & Frank, 2014). Thus, ICPS is often referred to as a measure of functional connectivity (e.g., Watts et al., 2018). Theta ICPS between medio-frontal and lateral-frontal sites indexes the degree to which detection of the error and need for cognitive control (i.e., by the ACC/ medial prefrontal cortex (PFC)) is communicated to relevant areas for instantiation of control (i.e., dorsolateral PFC; Buzzell et al.,

2019). The lateral prefrontal cortex (lPFC) is thought to execute the changes needed to achieve more optimal outcomes as specified by the cognitive control signal (Shenhav et al., 2013). The lPFC maintains task-relevant representations and recruits other neural areas relevant to executing and maintaining the cognitive control signal (Shenhav et al., 2013). In youth, dorsolateral and ventrolateral PFC activity has been associated with increased working memory ability (Crone & Steinbeis, 2017; Luna et al., 2010). Therefore, lPFC activity considered in tandem with ACC activity more fully represents the CC/EC mechanisms involved in anxiety.

In youth, preliminary evidence demonstrated that theta ICPS between medio-frontal and lateral-frontal sites reflects engagement in CC through examining its associations with task behavior and other TF metrics in tandem (Buzzell et al., 2019). To my knowledge, only one study has examined the developmental trajectory of theta ICPS and did not find significant effects of age in young children (Morales et al., 2022). However, given this study had a restricted age range, it may be that developmentally-dependent changes in theta ICPS occur in a more protracted manner and were thus not captured in this restricted age sample.

TF Analyses and Anxiety

Emerging work has examined how anxiety relates to theta TF measures in children and adults. In adults, one study found that individuals with a GAD diagnosis demonstrated increased total power after errors compared to healthy controls (Cavanagh et al., 2017). However, ITPS was not found to be associated with GAD diagnosis (Cavanagh et al., 2017). Two adult studies examined how worry relates ICPS between mediofrontal and frontal-lateral cites in all-female samples (Cavanagh et al., 2017; Moran et al., 2015). The studies had conflicting results, such that one study found that greater worry was associated with reduced ICPS and the other reported

the opposite pattern (Cavanagh et al., 2017; Moran et al., 2015). Thus, the nature of the association between anxiety and theta TF analyses in adults remains unclear.

In youth, only one study examined anxiety and theta-based TF indices. No associations between anxious behaviors and total power after errors were identified in pre-school age children. However, the relationship between social withdrawal and the ERN amplitude was significantly moderated by total power, such that the ERN amplitude was only associated with social withdrawal when total power was low (Canen & Brooker, 2017). Canen & Brooker (2017) interpreted these findings as indicating anxiety is associated with ineffective signaling for greater CC, although the neurophysiological foundations for this claim are unclear and no theoretical basis for the moderation analysis performed was provided. No studies examined associations between ITPS or ICPS and anxiety in youth.

The Current Study & Hypotheses

The current dissertation aims to extend previous research by examining the association between anxiety and a variety of neurophysiological measures of CC/EC in children and adolescents. Using EEG and questionnaire data from a longitudinal study of children ages 3 – 17 years old, I considered how anxiety relates to the ERN amplitude and error-related theta TF indices recorded during a developmentally-appropriate Go/No-Go task. Specifically, I examined two symptom dimensions of anxiety previously found to relate to the ERN amplitude in youth: GAD and social anxiety symptoms. The longitudinal nature of the data allows for both the role of initial levels of anxiety as well as the change in anxiety over time to be evaluated. I also considered how aging moderates the association between anxiety and each measure of CC, given evidence that age moderates the association between anxiety and the ERN and that

developmental changes occur in theta TF measures. I examined task performance to contextualize the neurophysiological findings (as recommended by Schroder & Moser, 2014).

First, based on past work and developmental extensions of the CEMH, I hypothesize that the association between anxiety and the ERN amplitude will be moderated by age, such that the association between anxiety and an enlarged ERN amplitude will become stronger as children get older (**Hypothesis 1**). This increase in the association between anxiety and the ERN is thought to reflect compensatory effort related to CC. The expected association in younger youth is less clear given mixed literature. It may be that anxiety relates to a smaller ERN or may not be associated with the ERN in younger youth.

Hypotheses for theta TF analyses are more preliminary given limited previous work that has examined TF measures with anxiety. Given that the ERN is thought to be reflective of frontal midline theta activity (Cavanagh & Frank, 2014), my hypotheses for theta power mimic those for the ERN amplitude. That is, I would expect a stronger association between greater power and higher anxiety when children are older (**Hypothesis 2)**. However, results for power could differ between the types of power and/or from findings involving the ERN amplitude, because these metrics of power capture unique aspects of neural oscillations not reflected in the ERN amplitude. Thus, analyses for different types of power are exploratory in nature.

Because greater ITPS has been shown to contribute to a larger ERN amplitude (DuPuis et al., 2015), I hypothesize that anxiety will be related to increased ITPS. Similar to ERN and power hypotheses, I would also expect that this association will be moderated by age, such that associations between anxiety and ITPS will be stronger when children are older (**Hypothesis 3)**. Greater anxiety in older children is expected to prompt an increased call for CC resources, such that neural efficiency in the call for resources are likely improved with age. In contrast, neural

efficiency would be expected to be poorer in younger children, such that they cannot generate a sufficient neural signal to compensate for anxiety.

As previously mentioned, findings pertaining to anxiety and ICPS are mixed in adults. In the fMRI literature, one study of adolescents identified that increased anxiety symptoms were associated decreased functional connectivity between the salience network, which includes the dACC, and several areas of the PFC considered to be part of the executive functioning network (Geng et al., 2016). These results are similar to ICPS findings of Moran et al. (2015) in adult women, which demonstrated increased worry symptoms were related to reduced ICPS between medio-frontal and lateral-frontal sites. Therefore, I hypothesize that increased anxiety will be associated with decreased connectivity between medio-frontal and lateral-frontal sites in youth (**Hypothesis 4)**. It is more difficult to speculate the role of age in the association between anxiety and ICPS. Anxiety may relate to reduced ICPS between these regions irrespective of age. Alternatively, it could be that the association between anxiety and ICPS will be moderated by age. If age moderates the association between anxiety and ICPS, I expect that the association would be stronger as children get older. Because anxiety has not been found to influence the error-related signaling at frontocentral sites in younger children in some studies of the ERN, it may be that anxiety similarly does not strongly impact the connectivity between sites in younger children due to poorer instantiation of control. Notably, there has been heterogeneity in the regions identified as carrying out cognitive processes that may support the instantiation of cognitive control in young youth, such that regions other than lPFC have been found to relate to processes such as inhibition and switching (Crone & Steinbeis, 2017). It is not clear at this time if this is related to true developmental differences or if this is reflective of sample and methodological differences across studies (for reviews: Crone & Steinbeis, 2017; Luna et al.,

2015). Thus, ICPS between medio-frontal and lateral-frontal sites may not fully reflect CC in younger children, such that associations between anxiety and ICPS may not be present in younger youth.

Finally, I hypothesized that the association between anxiety and task performance (i.e., number of errors and reaction time on correct trials) would be stronger in younger children (**Hypothesis 5**). Under developmental extensions of the CEMH, younger children may be unable to compensate for the theorized cognitive load of anxiety like adults and older children due to lack of resources available (Moser, 2017). I therefore hypothesize that anxiety will be associated with more errors and slower performance when children are younger.

In sum, I expect that the associations between anxiety and EC/CC will vary with development. I expect a stronger association between anxiety and neural measures of CC (i.e., ERN amplitude, power, and ITPS) as youth get older. Similarly, I expect anxiety will relate to worse performance in younger youth. Anxiety may be uniquely associated with the functional communication of the cognitive control signal to areas of cognitive control instantiation (i.e., frontocentral to mediofrontal ICPS), such that it may be related poorer functional communication across development or this association may be restricted to older youth.

Methods

Study Overview

Data was collected as part of the Michigan Longitudinal Study at Michigan State University. An overview of data collection is represented in Figure 1. Parent questionnaires were completed at baseline and then annually for three years, including Revised Child Anxiety and Depression Scale- Parent Form (RCADS-P) used to assess anxiety. Additionally, children attended three in-person visits across the three-year time period during which they completed the Go/No-Go Task for neurophysiological assessment. The original design included a baseline visit and 18- and 36- month (3 year) follow-ups.

Participants

A total of 236 children (125 Female, 111 Male) between the ages of three and 13 were recruited to participate in the current study at baseline. Multiple children from the same family were sometimes recruited for the study, resulting in a total of 139 families being included in the study. Ninety children (38.1%) were recruited from the Michigan Longitudinal Study, a multigenerational study in mid-Michigan examining neuroliabilities associated with risk of substance use disorders (Zucker et al., 2000; Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996). An additional 146 children (61.9%) were recruited from the community through online and paper advertising (Craigslist, Facebook, and community bulletin boards). Children from families living in any of the four counties surrounding the greater Lansing area in Michigan (Ingham, Shiawassee, Eaton, or Clinton county) were eligible if they were between the ages of three and 13 years old at enrollment. Children were also screened for neurophysiological testing eligibility which ruled out serious cognitive disabilities, autism spectrum disorder, epilepsy, head

trauma, and medical/visual/hearing issues that would affect their ability to perform computer tasks.

Procedure

Parents or legal guardians of eligible children received a consent form detailing study procedures, risks and benefits. After their parents/ guardians provided written consent, children ages eight to 13 were provided with a written assent in the laboratory, and children under the age of seven received a verbal description of the procedures during the laboratory visit.

Questionnaires were either mailed to participants or were completed via Qualtrics online. Parents completed a series of questions about themselves and their children before their child's scheduled neurophysiological visit, or they finished the questionnaires in the laboratory while the children completed the neurophysiological portion of the study. At each visit, experimenters explained each step of EEG set-up to the children. During all visits, participants wore an EEG cap and face sensors during a series of three tasks. An experimenter also remained present in the room during each task to give instructions, task-relevant reminders, and manage behavior as needed. Parents were permitted to stay in the room to observe or stay in a waiting room based on child needs. Participants completed a series of three developmentally-tailored tasks: flanker task, go/no-go task (the Zoo Game), a reward task (Doors task), and an emotion-modulated startle task. Following completion of EEG tasks, participants completed executive functioning/temperamental tasks. *The Zoo Game is the focus of the current dissertation.*

Parents received $50 for each questionnaire completed about their children. Children received $50 for the baseline EEG visit and $75 for each subsequent EEG visit. Children older than 8 years old received an additional $7.50 for completion of the reward task and children 10+ received $3.00 for completing the flanker task and Zoo Game.

Revised Child Anxiety and Depression Scale (RCADS)- Parent Report

The RCADS – Parent Report is a questionnaire containing 47 questions used to assess for dimensional symptoms of anxiety and depression. Parents responded to each item using a 4-point Likert scale (0 = Never, 1 = Sometimes, 2 = Often, 4 = Always). The scale includes six subscales (Separation Anxiety Disorder, Social Phobia, Generalized Anxiety Disorder, Obsessive Compulsive Disorder, Panic, and Major Depressive Disorder). Two total scores can be produced: one that includes the sum of all items for an overall index of anxiety and depression and the other that sums only the anxiety-related items for an overall anxiety index. Subscale and total scores were calculated by averaging responses across items. The scale was initially intended for use with children grades 3 – 12 and was demonstrated to have adequate reliability and validity (Ebesutani et al., 2010). However, acceptable reliability and validity was recently demonstrated within a sample of 3 - 17.5 year old children (Ebesutani et al., 2015). *Only the Generalized Anxiety Disorder and Social Phobia subscales were be used in the current analyses* (GAD Subscale: Baseline *Chronbach's α* = 0.85, Follow-Up Year 1 *Chronbach's α* = 0.84, Follow-Up Year 2 *Chronbach's α* = 0.80, Follow-Up Year 3 *Chronbach's α* = 0.87, Average *Chronbach's α* = 0.85 across timepoints; Social Phobia Subscale: Baseline *Chronbach's α* = 0.88, Follow-Up Year 1 *Chronbach's α* = 0.86, Follow-Up Year 2 *Chronbach's α* = 0.88, Follow-Up Year 3 *Chronbach's α* = 0.89, Average *Chronbach's α* = 0.88 across timepoints).

While baseline and 36-month follow-up visits correspond with times at which the RCADS-P was completed, the RCADS-P was not completed at the time of 18-month follow-up (see Figure 1). Precise dates of collection were not available questionnaires due to a collection error. Of the 189 total participants with a questionnaire completed at Year 1 or Year 2, 127 participants had data from both timepoints, 47 had data only available from Year 1, and 15

participants had data available from only Year 2. Therefore, if participants had useable RCADS-P data from both Year 1 and 2, I used use an average of the two RCADS subscale scores. If only one RCADS-P was available (it was either completed at Year 1 or Year 2), I used the available questionnaire.

The Zoo Game

Children completed a developmentally-appropriate Go/No-Go task adapted for EEG called the Zoo Game (Grammer, Carrasco, Gehring, & Morrison, 2014). Children were instructed to "capture" escaped zoo animals by pressing the spacebar quickly each time a zoo animal (Go stimuli) was presented on the screen. However, there were three orangutans (No-Go stimuli) that the children were specifically asked not to "capture" by inhibiting their response to press the spacebar. Before starting the task, children completed a practice block consisting of 12 trials: 9 with zoo animals other than orangutans and 3 with orangutans. The children then completed 8 blocks of 40 trials (each trial including 10 images of the orangutans and 30 novel zoo animal pictures), for a total of 320 trials. Each animal was presented on the screen for a maximum of 750 ms followed by a fixation cross (+) displayed for a randomized interval ranging between 200 and 300 ms/blank screen for 500 ms.. The image displayed disappeared once a response was made. Responses that occurred between 200ms and 1350ms were included in data analysis. The task lasted approximately 20 minutes. Stickers were given at block breaks as a reward for task completion. *No-Go Error Trials were the focus of the majority of analyses. Reaction times on Go Correct trials were also considered.*

EEG Recording

An Active Two Biosemi System (BioSemi, Amsterdam, The Netherlands) was utilized to obtain electroencephalogram (EEG) data using 64 Ag-AgCl electrodes placed in a stretch-lycra

cap in accordance with the 10/20 system as shown in Figure 2. The "10-20 system" refers to the standardized method of placing each of the scalp electrodes – each electrode is spaced apart from adjacent electrodes at a distance of either 10% or 20% of the total front-back to right-left distance of the skull. Measurements were taken to ensure proper cap fit, with cap size determined by the distance between the nasion (the distinctly depressed area between the eyes) and the inion (the lowest point of the skull on the back of the skull identified by a prominent bump). Centering of the cap was achieved by measuring the distance between the ears around the top of the head, with the tip of each ear being used as a measurement endpoint. A chin strap was used to hold the cap in place in a tight, but comfortable fashion. Electrodes were placed into each of the labeled ports, with labels consisting of combinations of letters and digits (e.g. Pz, C2, T7). The first letter of the label corresponds to areas of the cerebral cortex (i.e. F = frontal, T= temporal, C= central, P = parietal, and O = occipital lobes). The second part of the label can either be a letter or number and indicates location on the scalp in relation to midline sites. The letter "z" indicates a location along the midline of the scalp, while odd numbers indicate left hemisphere sites and even numbers indicate right hemisphere sites.

Sensors were also placed on the left and right outer canthi (the outer corners of the eyes where the upper and lower lids meet) and below the left eye (approximately 1cm from the pupil) to measure eye movements. Together with the FP1 headcap site, the eye sensors allowed us to remove electrooculogram (EOG) activity resulting from blinks and eye-movements that otherwise confound EEG activity. Two sensors were also placed on the left and right mastoids – bone protrusions behind the ears – to use during offline analyses as references. The Common Mode Sense (CMS) active electrode and the Driven Right Leg (DRL) passive electrode formed the electrical ground during data acquisition. In addition to acting as a reference, the CMS-DRL

loop ensures that the average voltage of the participant stays within a reasonable range, thereby limiting current that could potentially return to the participant. All signals were digitized at 1,024Hz, which represents 1,024 samples of data taken per second that provides millisecond precision.

EEG Processing

Overview

An overview of EEG processing is provided in Figure 3. First, data was preprocessed to remove artifacts and noise using a custom MATLAB (The Math Works inc.) script set containing both original and EEGLAB (Delorme & Makeig, 2004) functions. Briefly, processing steps included computing the average amplitude of the ERN, determining the frequency range within the theta band and time range for TF analyses, and calculating power, ITPS and ICPS via the Psychophysiological Toolbox (Bernat et al., 2005).

Preprocessing

Only participants with a no-go error rate of less than 60% were preprocessed and included in analyses, because such participants had performance that fell two standard deviations below the mean performance at baseline. A band-pass filter with cutoffs of 0.1 and 30Hz (12 dB/oct rolloff) was applied to the continuous data to remove extreme high and low frequency artifacts. The data was then resampled to 256Hz during preprocessing (data is later resampled down to 128Hz prior to computing the ERN amplitude and down to 32Hz for all TF analyses) for ease of processing. All trials were then corrected for eye movements and blinks using methods developed by Gratton, Coles, & Donchin (1983). Trials with reaction times that occurred outside of a 200 – 1300ms post-response window were removed from analysis. Then, three second epochs were created beginning 1000ms pre-response and ending 2000ms post-response to create

response-locked epochs. Steps were then taken to remove or clean data that contained activity suspected to reflect noise from sources other than the brain. Specifically, for each individual, trial epochs were ranked according to number of extreme (> ±150mV) data points across all channels, and the worst 5% of epochs were removed. Additionally, individual channels were interpolated across all data if they exceeded the threshold of 5 standard deviations in the domains of kurtosis and activity probability. After baseline (-200 to 0ms pre-response) correction occurred, each trial epoch was evaluated separately and channels with extreme (> ±150mV) data points were interpolated only for that epoch, while trial epochs with more than 2 bad channels were rejected and removed from the data. A final visual inspection was conducted to remove epochs with unusual artifacts.

Only participants who had usable data for at least four no-go error trials were included in analyses, which is generally considered acceptable for TF analyses given subsampling.

Subsampling

EEG metrics are affected by the number of trials used in their calculation (Buzzell et al., 2019; Fischer et al., 2017). To account for the effect of the number of trials available, I used subsampling methods described by Buzzell et al. (2019). For each participant, a random subsample containing 4 unique trials was selected. Then, each EEG metric of interest (i.e., ERN, power, ITPS and ICPS) was calculated for the subsample of trials. For each participant, this process was repeated 25 times, such that each participant was 25 estimates of the EEG metric. Finally, an average of the 25 estimates of EEG metrics was taken and used as the final estimate of the EEG metric for that participant.

ERN

The time-domain ERN amplitude was defined as the average amplitude of the negative deflection of voltage from 0 to 100 msec. The ERN amplitude was examined at the midline site where it is maximal (i.e., sites Fz, FCz, Cz, CPz, Pz). The average ERN amplitude for each participant was calculated using the subsampling and bootstrapping methods as described previously.

Cohen's class RID and TF Principal Component Analysis

To conduct TF analyses, first a TF decomposition of the average EEG activity on no-go error trials was created for each participant. The process was that detailed by Watts, Tootell, Fix, Aviyente, & Bernat (2018). First, 3rd order Butterworth filters were used to isolate theta frequency ranges. The frequency range for filters were selected based on visual inspection of unfiltered TF energy after an error is made for a 1s period. Next, the data was transformed from the time domain to the TF domain. TF transforms were created using a binomial reduced interference distribution (RID) variant of Cohen's class of TF transformations using the full epoch. The result is a TF-decomposed surface for the average EEG activity on no-go error trials (epoched from 1000ms – 2000ms) for each participant. In the TF-decomposed surface, time, frequency and power are each represented as three unique dimensions of the data.

To isolate the precise time range and frequency range of interest to capture post-error activity, a principal component analysis (PCA) was applied to the TF decomposed surface to identify the portions of activity of interest (Bernat et al., 2005). PCA is a feature detection technique that identifies components of activity that are meaningful while reducing the complexity/amount of TF decomposed surface that needs to be examined. The TF-decomposed surface for all participants and channels undergo PCA simultaneously and solutions were

evaluated for 1 – 6-component solutions. A scree plot of eigenvalues were used to select the appropriate PCA solution across participants. The component in the PCA solution occurring approximately within the time window of the ERN (0 – 100ms) was used in analyses.

Theta Evoked Power and Total Power

There are two measures of theta power that can be computed following processes detailed by Buzzell et al. (2019). Evoked power on error trials involves primarily phase-locked information that is computed from TF transformed data that has already been averaged across error trials (Buzzell et al., 2019). To compute evoked power, the factor loadings from the PCA solution were applied to the average TF data on error trials to create PC-weighted post-error evoked power for all channels and participants (Buzzell et al., 2019).

Total power includes phase- and non-phase-locked information and is computed from TF transforms of trial-level data (using TF transforms resutling from the RID as previously described; Buzzell et al., 2019). The TF-transformed data at the trial level were averaged for each participant (Buzzell et al., 2019). Factor loadings from the PCA solution were applied to the averaged trial level TF data (Buzzell et al., 2019). Sites were selected based on where the signal was maximal across midline sites.

Theta ITPS

Average theta ITPS was computed as specified by Watts et al. (2018). Phase locking values (PLVs) were calculated, which represent the average difference in phase synchrony between no-go error trials at a single site. PLVs were available for each channel within participant across theta frequencies. Mirroring power analyses, the same PCA solution factor loadings will then be applied to the ITPS surface to isolate ITPS within the time and frequencies of interest. The same site(s) used for power measures were used for ITPS analyses.

Theta ICPS between Medio-frontal and Lateral-frontal Sites

To assess functional connectivity between medio-frontal and lateral-frontal areas, theta ICPS was calculated between FCz and F3, FCz and F4, FCz and F5, and FCz and F6 (see Figure 3 for site locations; Moran et al., 2015). Theta ICPS analyses followed methods detailed by Watts et al. (2018). ICPS was calculated through phase synchrony computation based on Cohen's class of TF distributions (Aviyente et al., 2011). Data were transformed using current source density (CSD), which allows for activity to be localized to the cortical surface. Then, PLVs were calculated, which represents the average difference in phase synchrony between sites across epochs. Again, the PCA solution factor loadings from power analyses were applied to the ICPS analyses to isolate ICPS within the time and frequency ranges of interest.

Analysis Plan

To examine the associations between anxiety and EC/CC, a series of multilevel models were executed. Multilevel modeling can account for the repeated-measures nature of the data within participants and for some participants being from the same families. Further, multilevel modeling is flexible, as it allows for missing data within participants, such that participants can be retained in analyses even if they did not attend all visits.

Separate models were conducted to examine between-person differences at baseline and the within-person effect of change over time across observations. For all models, the following dependent variables were examined: number of no-go errors made, the ERN, evoked power, total power, ITSP, ICPS between FCz and F3, ICPS between FCz and F4, ICPS between FCz and F5, and ICPS between FCz and F6. To make effect size estimates more interpretable, ITPS and ICPS values were scaled by a factor of 1000 due to their small size.

Baseline/Between-Person Models

Fixed Effects Structure. The fixed effects structure allows for the association between anxiety and each measure of CC and its moderation by age to be examined. For baseline models, fixed effects included baseline/between-person age, baseline/between-person anxiety and their interaction. Baseline age and anxiety were grand-mean centered. Separate models were executed for social anxiety and GAD symptoms, resulting in two models being executed for each dependent variable.

Random Effects Structure. Multilevel modeling with two levels (i.e., individual and family) was conducted with a random intercept for family to account for dependence related to siblings being included in the sample.

Model Formula.

The formula for the model is as follows:

Level 1: $\beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_1 X_2 + \varepsilon_{ijk}$

Level 2: $\beta_0 = f_{00k}$

where the variance of f_{00k} represents the difference in the dependent variable between families, X_1 represents baseline age and X_2 represents baseline anxiety.

Longitudinal Models

Fixed Effects Structure. To account for between-person differences in age and within-person aging of participants over time, two variables were created and included for age. First, to account for aging over time, a person-centered variable was created that represents the change in age from the baseline visit and was calculated by subtracting age at baseline from age at the other time points. Second, a between-person variable was included that represents each participant's age at baseline and was grand-mean centered. For anxiety, I was interested in the

effect of the change in anxiety over time. Therefore, similarly a within-person anxiety variable was created to represent change in anxiety from the baseline visit, as was created for age.

The primary effects of interest in the longitudinal models involve within-person age and within-person anxiety. The model therefore included a 2-way interaction between within-person age and within-person anxiety. Additionally, between-person age at baseline was included as a main effect to account for the effect of initial age differences between participants on each CC dependent variable. Separate models were executed for social anxiety and GAD symptoms, resulting in two models being executed for each dependent variable

Random Effects Structure. Multilevel modeling with three levels were used to account for the repeated-measures: visit, individual and family. First, a random intercept was included for each participant to reflect that participants complete multiple visits over time. Additionally, a random intercept was included for family to account for dependence related to siblings being included in the sample. A random slope for age could not be estimated due to the low number of participants with more than 2 observations.

Model Formula.

The formula for the model is as follows:

Level 1: $\beta_0 + \beta_1 X_1 + \beta_2 X_2 + \beta_3 X_1 X_2 + \varepsilon_{ijk}$

Level 2: $\beta_0 = \gamma_{00} + \gamma_{01} X_3 + p_{0j} + \theta_{00}$

$\beta_1 = \gamma_{10}$

$\beta_2 = \gamma_{20}$

$\beta_3 = \gamma_{30}$

Level 3: $\theta_{00} = f_{00k}$

where the variance of p_{0j} represents the difference in the dependent variable between participants, the variance of f_{00k} represents the difference in the dependent variable between families, X_1 represents within-person Age, X_2 represented within-person anxiety, and X_3 represents between-person/baseline age

Sensitivity Analysis. To determine the size of the effect we were able to detect with the current sample, a sensitivity analysis was conducted using the G-power program with a repeated measures design as a proxy for MLM (for baseline models: number of subjects with complete data = 168; number of repeated measures = 2 for average number of kids per family; for longitudinal models: number of subjects included = 168; number of repeated measures = 3 for number of timepoints). The alpha probability level was set to .05 and the power probability was set to .8 to determine the expected effect size of a between-person interaction at 80% power. For baseline models, I estimated small correlations between dependent variables based on low ICC values for the random intercept for family in previously conducted analyses of the ERN (Gloe et al., under review, *r*s = 0.01 - 0.3). For longitudinal models, I used the average correlations across the three timepoints for each dependent variable for participants who completed all study timepoints ($r_{ERN} = 0.625$, $r_{EvokedPower} = 0.784$, $r_{TotalPower} = 0.531$, $r_{ITPS} = 0.651$, $r_{ICPS} = 0.228$, $r_{errors} = 0.495$, $r_{RT} = 0.698$). Results revealed we were adequately powered to detect small effects for all analyses ($\eta^2_{Baseline} = 0.023 - 0.030$, $\eta^2_{ERN} = 0.007$, $\eta^2_{EvokedPower} = 0.004$, $\eta^2_{TotalPower} = 0.009$, $\eta^2_{ITPS} = 0.007$, $\eta^2_{ICPS} = 0.015$, $\eta^2_{Errors} = 0.010$, $\eta^2_{RT} = 0.006$).

Results

Participants

A breakdown of recruitment, data-loss and sample size for each study time-point are provided in Figure 4. Two-hundred thirty-six children (124 Female, 113 Male) between the ages of three and 13 were recruited to participate in the Michigan Longitudinal Study (MLS) at Michigan State University. Of the 168 participants with usable data at baseline, 54% of children were male and 46% were female. Multiple children from the same family were recruited for the study, resulting in a total of 110 families being included in analysis at baseline. At baseline, Fifty-four percent of participants identified as White, 13% identified as multiracial, 5% of participants identified as Black and 1% of participants identified as Asian (27% of participant's mothers did not report their child's race). With regards to maternal highest level of education, 13% had a high school degree, 13% completed some college, 3% had a vocational tech degree, 13% had an Associate's degree, 21% had a Bachelor's degree, 9% had a Master's degree, 1% had a Doctoral, PhD, MD, JD or other advanced degree, and 5% endorsed other degree achievement (23% did not report their highest level of education). Nineteen percent of participant's mothers reported an annual income of less than $10,000, 13% reported annual income between $10,000 and $20,000, 7% reported annual income between $20,000 and $30,000, 18% reported annual income between $30,000 and $50,000, 16% reported annual income between $50,000 and $75,000, and 4% reported annual income above $75,000 (23% did not report their annual income). Thirty-eight percent of participants were a part of the original MLS sample, whereas 62% were recruited from the community.

The average age was 9.252 years (*SD* = 2.407) at baseline, 10.108 years (*SD* =2.534) at 18-month follow-up, and 10.961 years (*SD* = 2.645) at 36-month follow-up. Average change in

age from baseline was 1.717 years (*SD* = 0.406) at 18-month follow-up and 3.106 years (*SD* = 0.222) at 36-month follow-up. The age distribution at each timepoint can be found in Figure 5.

PC Solution

The scree plot resulting from the principle component analysis conducted on evoked theta power values across the three study timepoints (i.e., baseline, 18-month follow-up and 36-month follow-up) is shown in Figure 6. A 2-factor PC solution was selected based on the scree plot and because the 2-factor PC-solution for evoked power with similar time window as the ERN had a stronger correlation with the ERN amplitude (2-factor solution PC: r = -0.559, 3-factor solution PC: r = -0.524). The resulting PC solution is displayed in Figure 7. The first component was selected given similar time window to the ERN and used in all time-frequency analyses.

Descriptive Statistics

Descriptive statistics for each dependent variable can be found in Table 1. As reported by Gloe et al. (in preparation), there is a significant difference between the ERN and the correct-related negativity (CRN) at baseline (see Figure 8). The grand average waveform and topographic map of post-error activity across timepoints is also displayed in Figure 9. The PC-filtered total theta power is depicted in Figure 10. Correlations between dependent variables are available in Table A1 and described in the Appendix. Correlations between anxiety and age are also described in the Appendix.

Hypothesis 1 Results: ERN Models

Baseline Models

As demonstrated in Table 2 and consistent with expectations, older age at baseline was associated with a significantly more negative ERN amplitude at baseline (η^2 = 0.004). However,

GAD and social anxiety symptoms were not significantly related to the ERN amplitude, and this relationship was not significantly moderated by age. [1]

Longitudinal Models

As depicted in Table 3, aging from baseline to follow-ups was not significantly associated with the ERN amplitude. Unexpectedly, change in GAD and social anxiety symptoms were not significantly related to the ERN amplitude, and this relationship was not significantly moderated by aging.

Hypothesis 2 Results: Theta Power Models

Evoked Theta Power Models

Baseline Model. As shown in Table 4, older age at baseline was related to greater evoked theta power at baseline ($\eta^2 = 0.004$). Notably, change in GAD and social anxiety symptoms were not significantly related to evoked theta power, and this relationship was not significantly moderated by age.

Longitudinal Models. As seen in Table 5, aging from baseline to follow-ups was not significantly associated with evoked theta power. Additionally, contrary to expectations, change in GAD and social anxiety symptoms were not significantly related to evoked theta power and this relationship was not significantly moderated by aging.

Total Theta Power Models

Baseline Models. As demonstrated in Table 6 and contrary to expectations/hypotheses, baseline age, GAD symptoms, social anxiety symptoms were not significantly related to total

[1] Exploratory models were conducted for each set of models substituting a categorical age variable for continuously coded age. Based on assertions from Meyer (2018) that the association between anxiety and the ERN shifts between ages 8 – 9, age categories were under 7 yrs. ($N = 31$), 8 – 9 yrs. ($N = 75$), and older than 9 ($N = 62$). This categorical age variable was effects coded. Age moderation effect size estimates from models including categorical age did not differ from that of models including continuous age. Therefore, only results from models including continuous age are presented.

theta power at baseline. The interactions between baseline age and anxiety symptoms were also non-significant.

Longitudinal Models. As demonstrated in Table 7 and contrary to expectations/hypotheses, aging from baseline to follow-ups, change in GAD symptoms, change in social anxiety symptoms were not significantly related to total theta power at baseline. The interactions between aging and change in anxiety symptoms were also non-significant.

Hypothesis 3 Results: Theta ITPS Models

Baseline Models

As indicated in Table 8 and expected based on prior work, older age at baseline was associated with significantly greater theta ITPS ($\eta^2 = 0.009$). However, GAD and social anxiety symptoms were not significantly related to theta ITPS, and this relationship was not significantly moderated by baseline age.

Longitudinal Models

As shown in Table 9, aging between baseline and follow-ups was related to reduction in theta ITPS ($\eta^2 = 0.005$). However, change in GAD and social anxiety symptoms were not significantly related to theta ITPS and this relationship was not significantly moderated by aging.

Hypothesis 4 Results: Theta ICPS Models

Baseline Models

Results of baseline models with ICPS between FCz and left frontal sites (i.e., F3 and F5) as the dependent variable are shown in Table 10. In line with expectations, there was a significant interaction between GAD symptoms and baseline age ($\eta^2 = 0.009$). In breaking down this interaction, there was a significant association between GAD symptoms and ICPS between FCz and F3 only among youth under 4.91 years old ($p < 0.05$). Contrary to my hypotheses,

greater GAD symptoms predicted increased ICPS between FCz and F3 in youth under 4.91 years old (B = 0.113, SE = 0.058). Notably, this effect should be interpreted with caution given that only 8 children within our sample fall within this age range. No significant simple slope was identified in youth other than 4.91 years old ($p > 0.05$). SAD symptoms and its interaction with age did not relate to ICPS between FCz and F3. Also, baseline age, GAD symptoms, SAD symptoms were not significantly related to ICPS between FCz and F5, contrary to expectations.

Additionally, results of baseline models with ICPS between FCz and right frontal sites (i.e., F4 and F6) as the dependent variable are depicted in Table 11. Older age at baseline predicted strong connectivity between FCz and right frontal sites (η^2 = 0.005). However, contrary to hypotheses, GAD and SAD symptoms were not significantly associated with ICPS between FCz and right frontal sites, nor was there significant moderation of this association by baseline age.

Longitudinal Models

Results of the longitudinal models with ICPS between FCz and left fronal sites (i.e., F3 and F5) as the dependent variable are shown in Table 12. Recruitment group significantly predicted ICPS between FCz and F5, such that those in the original MLS sample had greater functional connectivity between sites (η^2 = 0.009). Aging, change in GAD symptoms, change in SAD symptoms and their interactions were not significantly related to ICPS between FCz and left frontal sites, contrary to expectations.

Results of the longitudinal models with ICPS between FCz and right frontal sites (i.e., F4 and F6) as the dependent variable are depicted in Table 13. Contrary to hypotheses, change in GAD and SAD symptoms were not significantly associated with ICPS between FCz and right lateral frontal sites, nor was there significant moderation of this association by aging.

Hypothesis 5 Models: Task Performance (No Go Errors and Go Correct RT)

Number of No-Go Errors Models

Baseline Models. Results of baseline models with number of errors made as the dependent variable are shown in Table 14. As expected, older age at baseline was related to making significantly fewer no-go errors (η^2 = 0.013). Contrary to my hypotheses, GAD and social anxiety symptoms were not significantly associated with number of no-go errors made, nor was this association significantly moderated by age.

Longitudinal Models. Results of longitudinal models with number of errors made as the dependent variable are shown in Table 15. As expected, aging from baseline to follow-ups were related to making significantly fewer no-go errors (change in age: η^2 = 0.035). Being part of the original MLS sample was associated with making significantly more errors (η^2 = 0.017). However, contrary to my hypotheses, change in GAD and social anxiety symptoms were not significantly associated with number of no-go errors made, nor was this association significantly moderated by aging.

Reaction Time on Go Correct Trial Models

Baseline Models. As shown in Table 16 and in line with expectations, older age at baseline was associated with significantly faster reaction time on Go correct trials (η^2 = 0.040). Contrary to my hypotheses, GAD and social anxiety symptoms were not significantly associated with the reaction time on Go correct trials, nor was this association significantly moderated by age.

Longitudinal Models. As shown in Table 17 and in line with expectations, aging from baseline to follow-ups were associated with significantly faster reaction time on Go correct trials (change in age: η^2 = 0.058). Contrary to my hypotheses, change in GAD and social anxiety

symptoms were not significantly associated with the reaction time on Go correct trials, nor was this association significantly moderated by aging.

Discussion

The current study explored how age moderates the association between anxiety and various neurophysiological and behavioral measures of cognitive control in youth. Contrary to my hypotheses, anxiety was not related to task performance, the ERN, theta power and theta ITPS at baseline, nor were changes in anxiety related to cognitive control measures over time. Further, this association was not moderated by baseline age or aging for most cognitive control measures. Although GAD symptoms and baseline age significantly interacted to predict ICPS between FCz and F3, this association was only present in youth younger than age 4.9 years which represent a very small portion of our sample. Anxiety and age did not interact to predict other ICPS metrics.

What is the Nature of the Association between Anxiety and Cognitive Control in Youth?

The lack of associations between anxiety and cognitive control in the current study were inconsistent with extant theories (i.e., Moser, 2017; Meyer, 2017). However, null findings are present in the anxiety-ERN literature in youth (for discussion: Meyer, 2017). A recent review suggests that research examining anxiety and the ERN may suffer from the file-drawer problem (Saunders & Inzlicht, 2020), such that null findings may be more common than published literature suggests. Thus, the current findings are not without precedent.

Such null findings may be indicative of the nuanced nature of this association, such that anxiety only relates to cognitive control under certain context and/or in particular individuals. For example, the current design examined children across a wide range of baseline ages. The limitation of such a design is that I may have been underpowered to detect small effects occurring at a specific age or narrower range of ages.

There are also other moderators outside of age that may further alter this association. For instance, previous findings have indicated that sex moderates the association between anxiety and the ERN in adults (for review: Moser, Moran, Kneip, Schroder, & Larson, 2016) and preliminary evidence suggests this moderation may also be present in youth (Ip et al., 2019, but see also Gloe et al., in preparation). The current sample also differs from previous samples that have examined anxiety and the ERN in that a substantial portion of the sample was recruited because of family history of substance use disorder. It may be that risk factors associated with family history of substance use disorder could also affect the association between anxiety and cognitive control in unexplored ways. For example, youth with a family history of substance use disorder are more likely to have greater impulsivity and externalizing behaviors (e.g., Dougherty et al., 2015), which are uniquely related to the ERN in youth (Lo, 2018).

It may also be that stressors alter the association between anxiety and cognitive control. In particular, future work should examine how experiencing unique stressors related to socioeconomic status, race, ethnicity, sexual orientation, gender and ability status (i.e., discrimination, systemic bias) may inform how anxiety relates to cognitive control and youth. Indeed, others have suggested that individuals with larger ERN amplitudes may be more likely to develop anxiety after exposure to stressors (Weinberg et al., 2022). They suggest that those with an enlarged ERN may be more susceptible to negative consequences of stressors (Weinberg et al., 2022). Alternatively, stress-related worries may further usurp cognitive resources in anxious youth after exposure to stress, resulting in greater calls for compensatory effort and strengthening the association between anxiety and the ERN (Moser et al., 2013; Moser 2017).

Methodological factors may also alter the strength of the association between anxiety and cognitive control. For instance, previous studies of anxiety and the ERN in youth have used a

variety of tasks, including the Go-No Go, Flanker, and Stroop tasks to evoke the ERN. Within each of these task types, tasks can differ on whether performance-based feedback is provided, by the stimuli used (e.g., letters, arrows, pictures), and by how responses are made (e.g., with both hands or one hand; keyboard, button-box or mouse; Gloe & Louis, 2021). These differences may alter the magnitude of the association between anxiety and the ERN (Gloe & Louis, 2021). Past work has also differed in the type of anxiety examined and the measures used to assess for anxiety. Notably, the current study is only the second to use the RCADS-P to study the association between anxiety and neurophysiological measures of cognitive control (initial study to use RCADS-P: Lo et al., 2016).

Symptom type and severity may also play a role in the nature of the association between anxiety and cognitive control. While I examined symptoms dimensions that have been shown in prior to work to relate to the ERN (i.e., GAD symptoms and social anxiety symptoms), others have found associations between cognitive control and dimensions of anxious temperament or overall anxiety metrics (Meyer, 2017; Moser, 2017). Others have suggested that anxious temperament is essential to consider in how anxiety relates to cognitive control (for discussion: Barker, Buzzell, & Fox, 2019). Therefore, future work should consider how anxious temperament plays a role in this association. Some have also asserted that the ERN is associated with risk of anxiety disorder development, rather than simply being associated with symptom severity at a particular time (Weinberg et al., 2022). Thus, including family history of anxiety disorders in future work may explain important variance in the association between anxiety and cognitive control. It should also be consider that this relationship may only emerge when levels of anxiety are sufficiently high. In youth, the association between anxiety and the ERN has been more consistently identified in clinically anxious samples than in community samples (for

review: Meyer, 2017). In the current study, the community sample had relatively low levels of anxiety symptoms, with average item responses ranging between “none” and “sometimes”. Because norms are not available for children under grade 3, it is somewhat difficult to compare how levels of anxiety in our sample compare to previously conducted work. Examining descriptive statistics for anxiety for studies of anxiety and the ERN, anxiety levels seem heterogeneous from sample-to-sample, with some studies capturing a wider range of severity than others. It is possible this heterogeneity could explain varying direction and effect size of the association between anxiety and the ERN across the pediatric literature.

The current study is strong in its inclusion of multiple measures of cognitive control, including TF metrics that have rarely been investigated in the context of anxiety. However, there are other measures of cognitive control that could be considered in future work. For example, while the current study focused on post-error metrics, the N2 is a stimulus-locked ERP that has been considered as another metric of cognitive control/effortful control and should be considered in future work. Some reseach has suggested anxiety is related to a larger N2 in youth, although this literature is somewhat mixed (for review: Lo, 2018). Additionally, it has been proposed that anxiety may alter the time course of cognitive control engagement, such that anxious youth are more likely to recruit cognitive resources just as they are needed in response to environmental stimuli (i.e., reactive control style) as opposed to employing low levels of sustained cognitive resources to hold goal-directed objectives in mind (i.e., proactive control style; Braver, 2012; Moser et al.,, 2013). Some have suggested that proactive control can be index through post-error theta ICPS between mediolateral and frontal lateral sites (Buzzell et al., 2019) and that the ERN may reflect reactive control engagement (Moser et al., 2013). Therefore, our findings may indicate that anxiety does not relate to reactive or proactive control engagement. However, others

have suggested that neural indicators of reactive control engagement occur pre-response (Buzzell et al., 2019), which I did not consider in the current analysis. I also did not employ a commonly-used behavioral measure of proactive and reactive control in the current study (i.e., the AX-CPT task). Therefore, it is possible that anxiety may relate to other measures of proactive and reactive control. Several recent studies of youth have demonstrated that anxiety symptoms relates the time course of cognitive control engagement, although the nature of this association seems similarly nuanced such that it is important to consider child temperament and age (Filippi et al., 2022; Troller-Renfree et al., 2019; Valadez et al., 2022).

The role of motivation should also be considered in future work examining anxiety and cognitive control. Many have suggested that motivation and reward sensitivity play an important role in cognitive control (for review in adults: Botvinick & Braver, 2015; Yee & Braver, 2018, Gray & McNaughton, 2003; in adolescents: e.g., Luna et al., 2015; Romer et al., 2017). Some have proposed that that the dlPFC may be where the motivation and cognitive control systems interface, while others have suggested that the dACC may serve to integrate valuation information and use it for the implementation of control (for review: Botvinick & Braver, 2015; Luna et al., 2015). It has been proposed that motivation also plays a role in anxiety development (e.g., Gray & McNaughton, 2003; Weinberg et al. ,2022). Indeed, one theory suggests that adolescents with temperamental risk factor for anxiety development are at risk for social anxiety disorder development due to, in part, greater sensitivity to motivational goals that increase avoidance tendencies (Caouette & Guyer, 2014). More work is needed to consider how motivation may moderate the associations between anxiety and cognitive control in youth.

Notably, I did not identify longitudinal evidence for change in anxiety relating to cognitive control, nor did change in age moderate this association. In addition to the

aforementioned explanations, it is possible that change in anxiety only has significant relationships with the ERN and TF metrics when they occur over a longer period of time than observed in the current study (i.e., longer or more sustained changes than the 18-36 months follow-up periods). It is also possible that changes in anxiety are only meaningful during specific developmental stages, such that baseline age may be an additional moderator. A three-way interaction between baseline age, change in age and change in anxiety was not attempted difficulties with interpretation and power concerns related to testing a three-way interaction, but future work with larger samples should investigate this interaction. Similarly, it may be that changes in anxiety are only meaningful at certain levels of baseline anxiety. I did not test the three-way interaction between baseline anxiety, change in anxiety and change in age due to difficulty interpreting three-way interactions involving all continuous predictors and concerns about overfitting our data with such an interaction, but I encourage investigation in future work with larger samples.

Others have also suggested that the ERN is a risk factor for anxiety development (e.g., Meyer et al., 2021; Weinberg et al., 2022). Earlier/baseline cognitive control, rather than change in cognitive control, may be more important to consider in the association between anxiety and the ERN. Thus, perhaps future analyses might consider different directionality (i.e., cognitive control metrics as predictors of anxiety) and/or predicting follow-up cognitive control with baseline anxiety.

How Does Development relate to the ERN and TF-Metrics of Cognitive Control?

The current study is novel in its inclusion of TF metrics of cognitive control, which have only begun to be investigated in youth. While results with respect to anxiety were null, several notable developmental effects were identified with respect to the ERN and TF metrics. In line

with previous findings (DuPuis et al., 2015; Gavin et al., 2019), older age at baseline was associated with a larger ERN and greater ITPS. Further, I identified older age was related to greater theta evoked power, contributing to the mixed literature of associations between power and age (DuPuis et al., 2015; Gavin et al., 2019; Morales et al., 2022). Finally, older age at baseline related to greater ICPS between frotocentral and right frontolateral sites. These findings are indicative of greater, more efficient cognitive control ability at later stages of child development.

In line with preliminary work (Morales et al., 2022), we did not identify developmental changes in theta ICPS between frontocentral and left frontolateral sites. It is possible that the connectivity or communication between these sites is developmentally insensitive in a lateralized fashion. That is, the strength of connectivity remains the same throughout development while the relative ability of error-monitoring at frontocentral regions (e.g., the ACC) and the ability to engage in executive functioning processes implement changes (e.g., the prefrontal cortex) each develop to lead to more effective and efficient cognitive control. It is also possible, as with other findings more generally, that developmental changes occur at a particular time or stage of childhood development that we were underpowered to detect in the current analysis. Further exploration of lateralization is needed.

There were a number of developmental effects that were surprising. Baseline age did not relate to theta total power. The presence of a baseline age effect for evoked and not total power suggests that there may be developmental differences in the power of oscillations with consistent timing across trials, but not in the power of oscillations overall irrespective of signal consistency. Along with the findings that baseline age relates to greater ITPS, my findings contribute to extant

evidence that neural efficiency, rather than signal strength itself, is greater in older youth than younger youth (DuPuis et al., 2015).

Notably, as with anxiety-related effects, we failed to identify effects of aging for most metrics in our longitudinal models. As previously mentioned, it may be that the time between most participant's observations was simply too small to capture an effect. Importantly, aging was, counterintuitively, associated with *less* ITPS. It could be that aging has distinct effects depending on baseline age, such that an interaction between baseline age and aging is critical to consider in future work to better understand why aging demonstrated the opposite effect as baseline age in this analysis.

Limitations

The current study offers a strong test of previous theories of anxiety and cognitive control by employing a longitudinal design with a wide age range at baseline. However, in addition to aforementioned future directions, there are a few other important limitations to consider. First, the timing of questionnaire administration was not ideal, as questionnaires were not directly administered at 18-month follow-up. While I attempted to account for this design issue, my longitudinal results may have differed if questionnaire timing was more precise. Additionally, relatively few children completed 36-month follow-up visits, weakening our longitudinal models further. I also did not include other psychopathology symptoms in my analyses, such as externalizing or mood symptoms, and it is possible that controlling for such comorbidities may have bearing on our findings (for further discussion: Weinberg et al., 2022). This might be particularly important in the current study as the sample at risk for externalizing and mood as a function of family substance use history. With respect to the processing of EEG data, I chose to focus my analyses on No-Go error trials alone given statistical model complexity. However,

future work should consider examining the CRN and TF metrics for EEG data following Go correct trials and the difference between errors and corrects across measures. Finally, demographic information regarding SES and race was missing for about a quarter of our sample due to an oversight in design, making the composition of the sample less clear.

Conclusions

Despite my null findings, the association between anxiety and cognitive control remains an important topic of investigation. Understanding how cognitive control, and, in turn, self-regulation, informs and/or changes as a result of anxiety development may reveal critical intersections between clinical, cognitive and motivation systems throughout development. Continued investigation could also point to novel treatment targets, such as treatments that aim to foster cognitive control and self-regulation in youth. My findings contribute to a larger body of work suggesting that this association is nuanced and may emerge in specific contexts and for certain individuals. Given the known importance of individual differences in clinical science, it is perhaps unsurprising that the association between anxiety and cognitive control may differ based on other individual or group-level/systemic factors. Conducting more longitudinal work across the lifespan that considers a wide variety of anxiety and cognitive control measures with diverse samples will ultimately lead to knowledge more readily translated to real-life clinical contexts.

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APPENDIX

Figures

Figure 1. Diagram of the study design. Three EEG visits were completed at baseline, 18-month follow-up and 36-month follow-up. The Revised Child Anxiety and Depression Scale- Parent Report (RCADS -P) was administered at baseline, 12-month follow-up, 24-month follow-up, and 36-month follow-up, as indicated by stars. I plan to select the RCADS-P completed closest to the 18-month follow-up EEG visit.

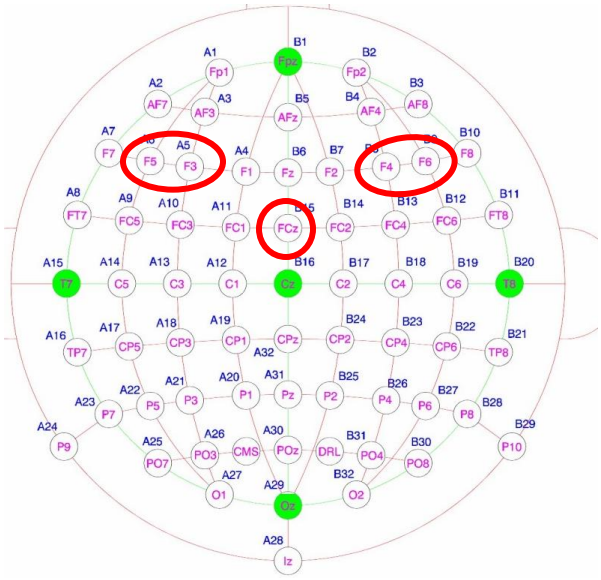


Figure 2. Head map of the BioSemi 64 electrode layout (*BioSemi Layout 64 + 2 Electrodes*, n.d.). Midline sites of interest for my analyses are Fz, FCz, Cz, CPz, and Pz. ICPS was conducted between sites FCz and F3, FCz and F4, FCz and F5, site FCz and F6 (circled in red).

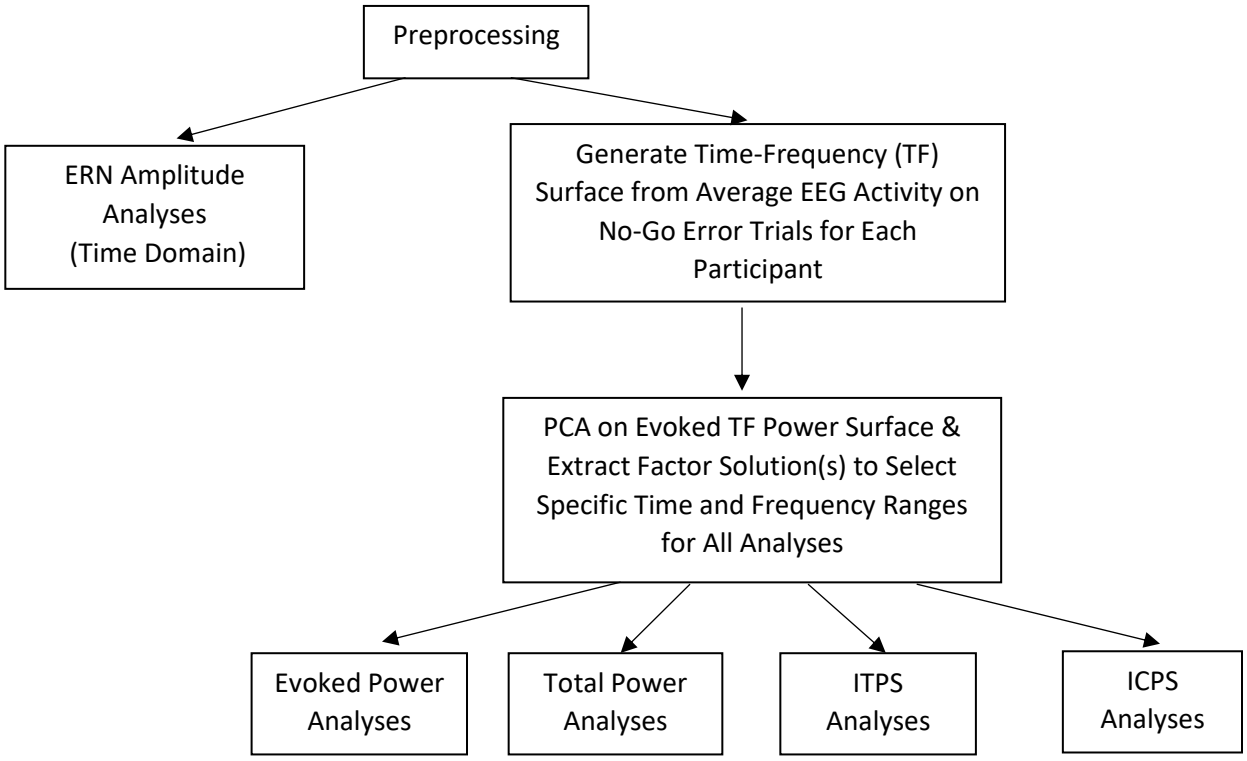


Figure 3. Overview of EEG Processing. Preprocessing of the data involves the removal of artifacts and noise such that brain activity is isolated. Error-related Negativity (ERN) analyses simply involve taking the average amplitude of EEG activity occurring after errors in the 0 – 100ms post-response window. For all other analyses, TF decomposition is performed on average EEG activity across no-go error trials for each participant. Principal component analyses (PCA) is used to extract the frequency and time ranges of interest and the PCA solution factor loadings are applied to each TF measure. Resampling is used in computation of all EEG metrics.

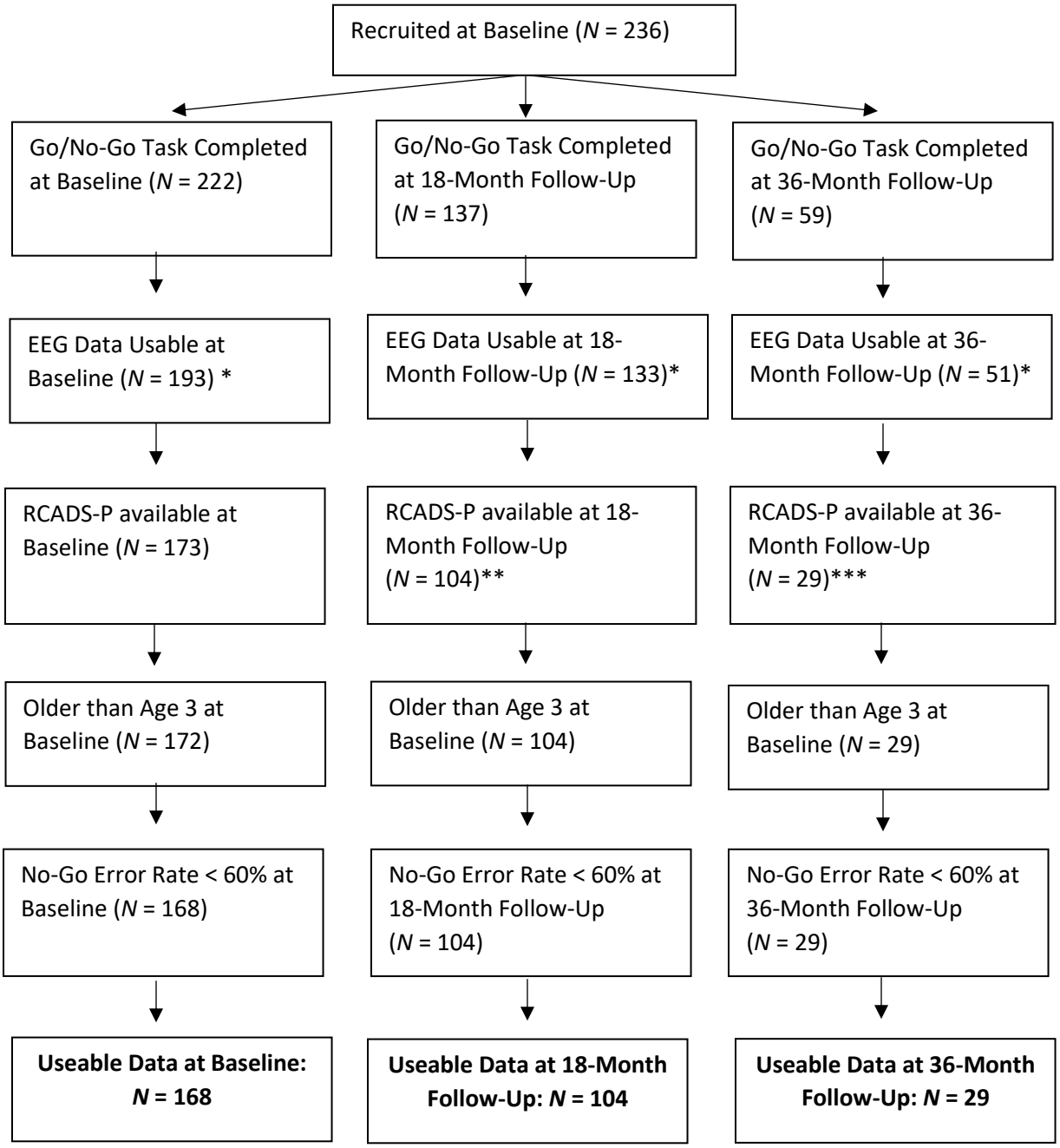
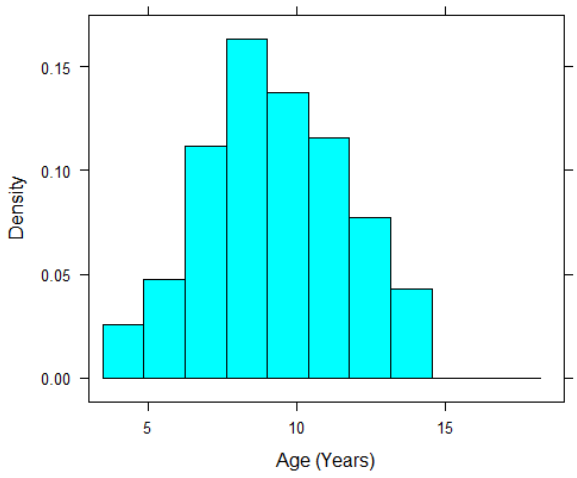


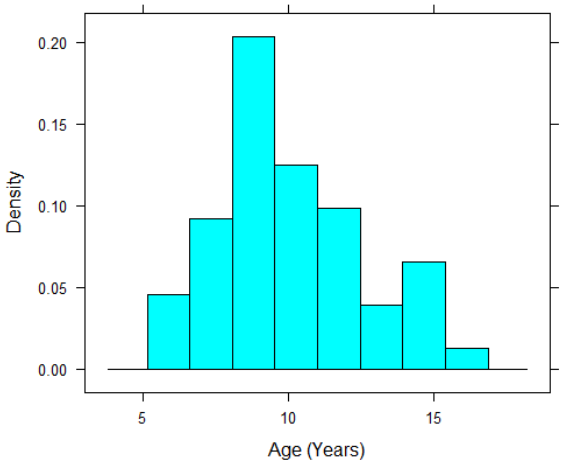
Figure 4. Illustration of data loss for each of the three study timepoints with final sample size utilized in data analysis. *Participants may have had EEG that was unusable due to data having excessive noise and/or failing to have at least four No-Go error trials with useable data (see Methods section for pre-processing description). **Participants had to have at least one complete

Figure 4 (cont'd)

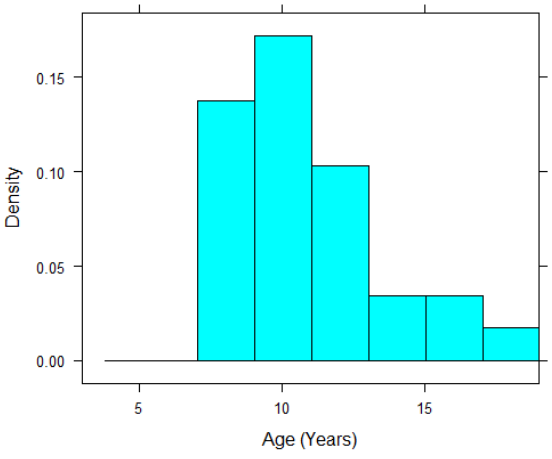
RCADS-P questionnaire at 1- or 2-year follow-up to be useable, as well as have a useable baseline RCADS-P questionnaire so a change score could be calculated. *** Participants had to have a complete RCADS-P at 36-month follow-up as well as have a useable baseline RCADS-P questionnaire so a change score could be calculated.



A)



B)



C)

Figure 5. Age distribution of participants at baseline (A), 18-month follow-up (B), and 36-month follow-up (C).

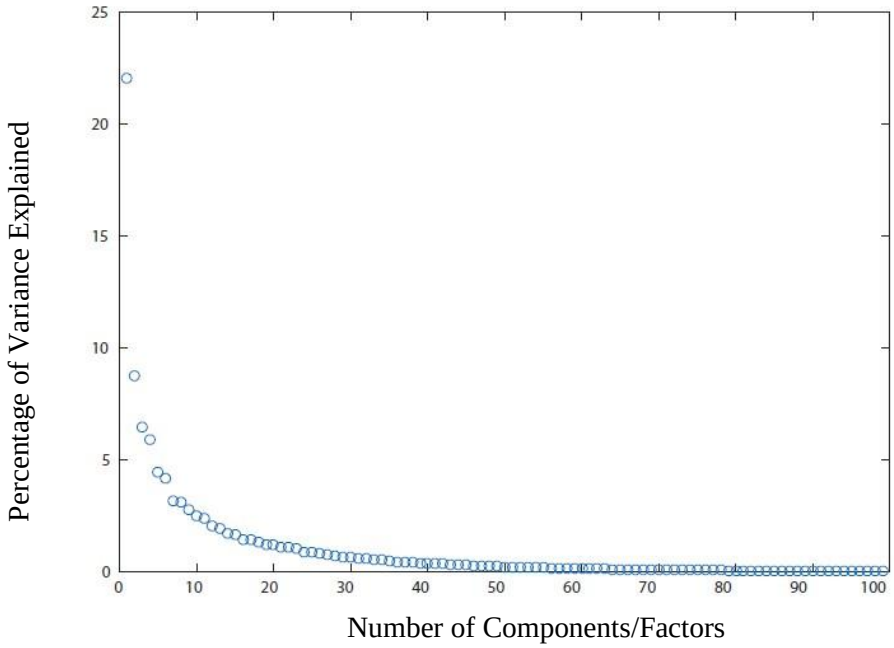
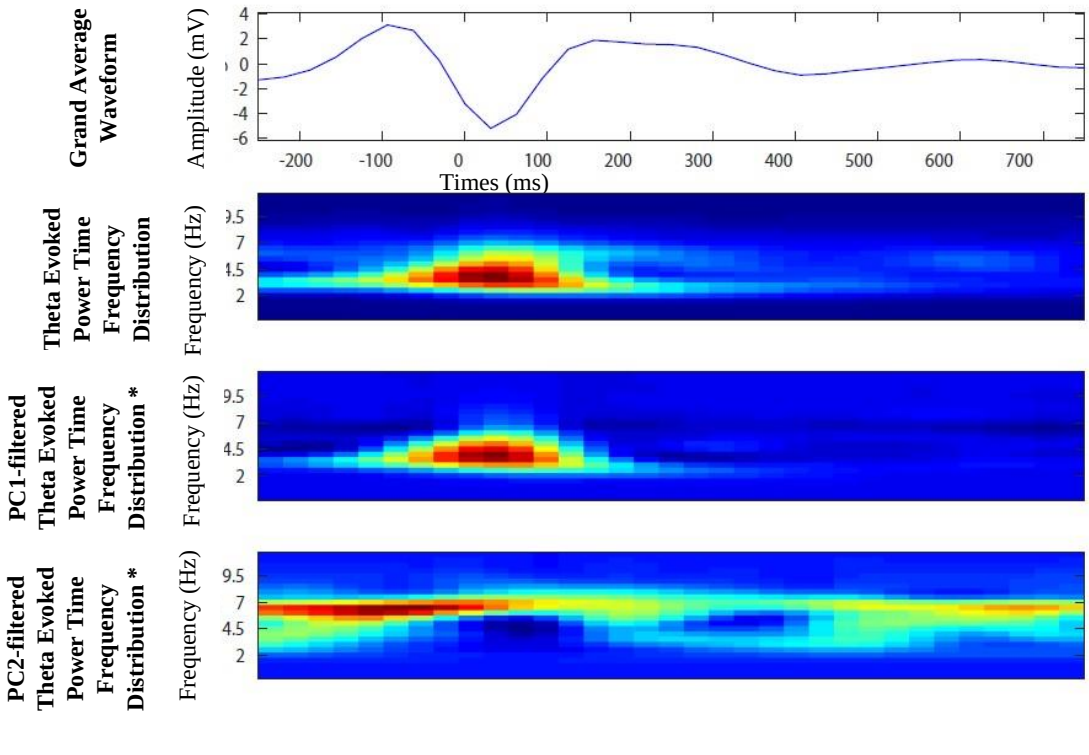


Figure 6. Scree plot resulting from principal component analysis of evoked theta power time frequency surface. A 2-factor solution was selected.



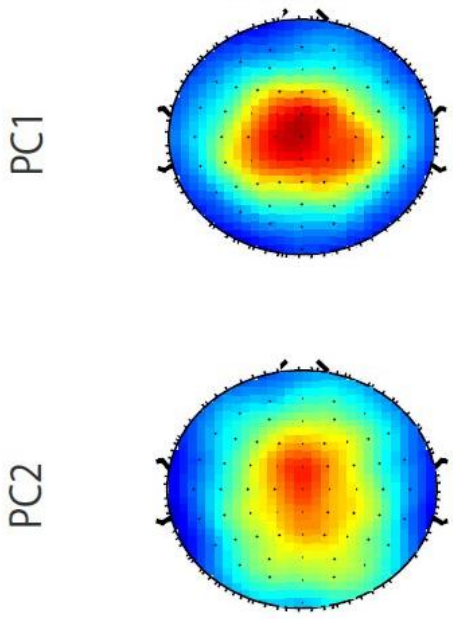


Figure 7. Two-factor principle component analysis solution for evoked theta power as (a) applied to the time-frequency distribution at FCz and (2) depicted on topographic maps of mean evoked theta power. Red indicates greater evoked theta power. PC1 (i.e., Principle Component 1)

Figure 7 (cont'd)

was utilized in all analyses given the similarity in its timing to the ERN. High levels of evoked theta power are seen centrally.

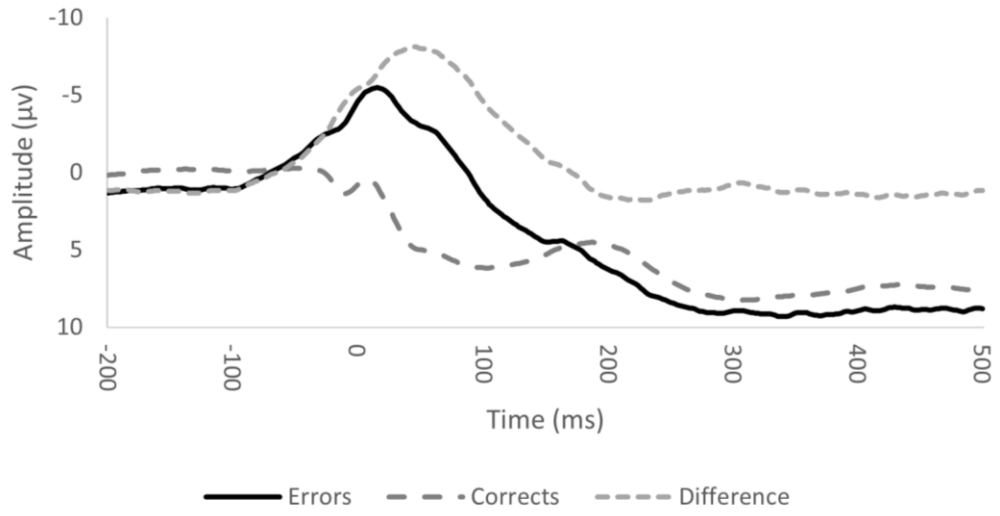
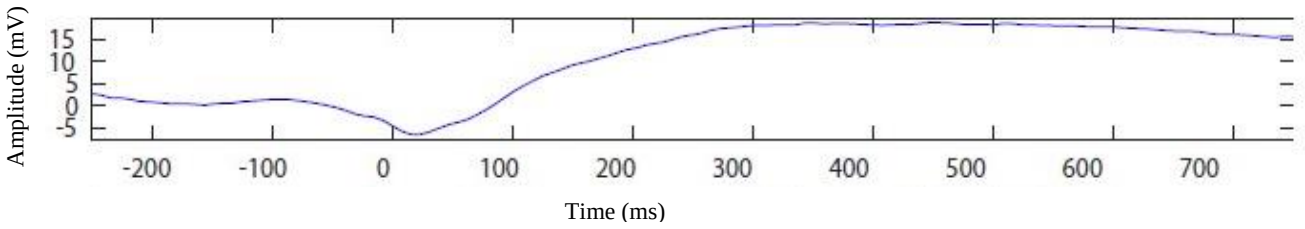
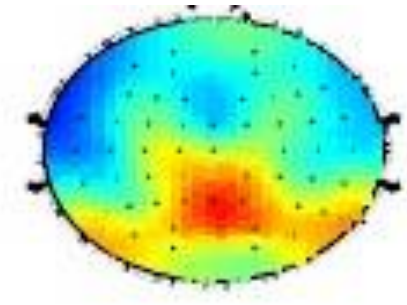


Figure 8. Grand average waveforms for post-error and post-correct activity pooled across FCz, Cz, Fz, FC1, and FC2 at baseline from Gloe, Sem, Winters, Durbin, & Moser (in preparation). There is a significant difference between the ERN and correct-related negativity (CRN) at baseline.

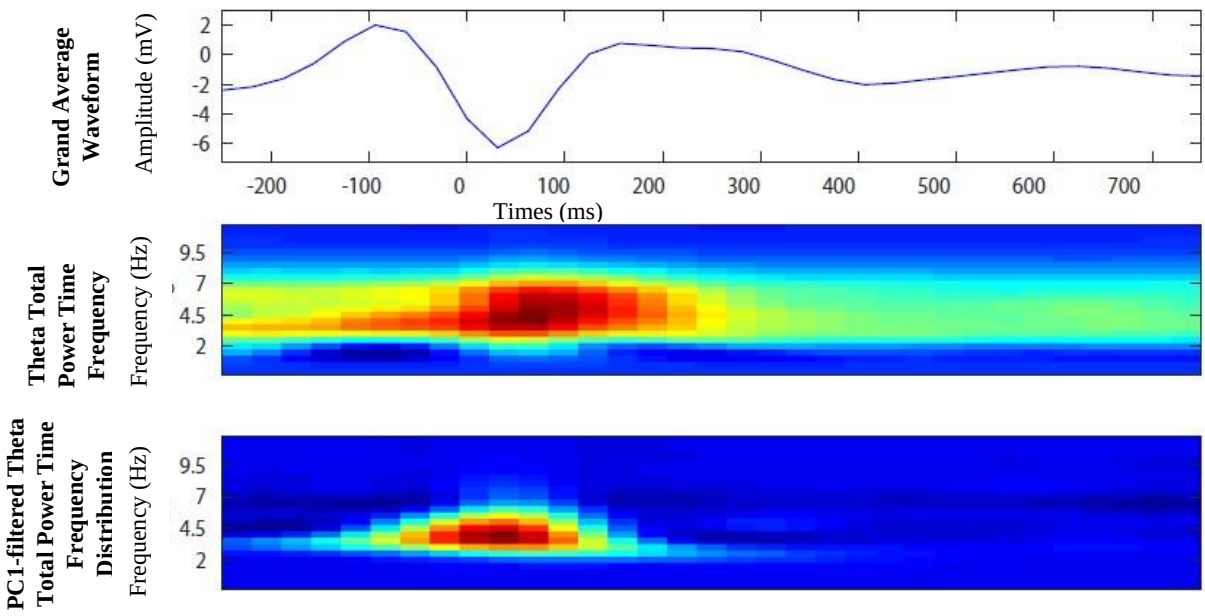


A)

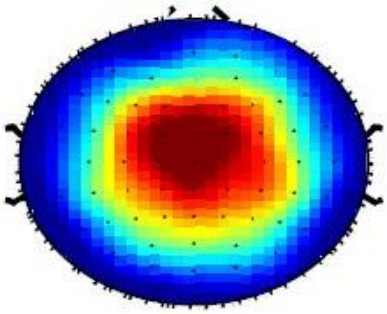


B)

Figure 9. Unfiltered error-related activity in the time domain. A) The grand average waveform for unfiltered post-error activity at FCz. The ERN occurs approximately between 0-100ms. B) A topopgraphic map of the average amplitude of the ERN between 0-100ms, with blue indicating more negative activity and red indicating more positive activity. The ERN occurs at frontocentral sites, denoted in blue on the topographic map.



A)



B)

Figure 10. (A) Theta total power unfiltered and PC1-filtered time-frequency distribution, with red indicating greater theta total power. B) Topographic map of PC1-filtered average total theta power, with red indicating greater theta total power. Highest levels of average theta power are seen centrally.

Tables

Table 1: *RCADS GAD Symptoms, Separation Anxiety Symptoms, Social Anxiety Symptoms, Task Performance and EEG Metrics Means and Standard Deviations*

RCADS Subscales

Measure	Mean (SD)	Minimum	Maximum
RCADS GAD Symptoms at Baseline (Item- Level Average)	0.556 (0.487)	0.000	3.000
RCADS Change in GAD Symptoms from Baseline (Person Centered)	0.048 (0.362)	-1.583	1.333
RCADS Social Anxiety Symptoms at Baseline	0.817 (0.574)	0.000	2.667
RCADS Change in Social Anxiety Symptoms from Baseline (Person Centered)	0.064 (0.417)	-1.389	1.444

Go/No-Go Errors

Measure	Mean (SD)	Minimum	Maximum
Number of No-Go Errors at Baseline	24.792 (9.562)	4.000	48.000
Number of No-Go Errors at 18-Month Follow-Up	21.798 (8.927)	5.000	46.000
Number of No-Go Errors at 36-Month Follow-Up	20.586 (7.209)	7.000	42.000

Go/No-Go Go Correct Reaction Time

Measure	Mean (SD)	Minimum	Maximum
Go Correct Reaction Time at Baseline	488.687 (70.090)	346.963	723.104
Go Correct Reaction Time at 18-Month Follow-Up	464.513 (66.316)	346.809	680.411
Go Correct Reaction Time at 36-Month Follow-Up	439.197 (52.249)	318.008	552.945

Error-Related Negativity (ERN) Amplitude

Measure	Mean (SD)	Minimum	Maximum

Table 1 (cont'd)

ERN Amplitude (mV) at Baseline	-4.404 (7.896)	-33.398	28.490
ERN Amplitude (mV) at 18-Month Follow-Up	-3.871 (7.236)	-33.612	19.847
ERN Amplitude (mV) at 36-Month Follow-Up	0.055 (6.038)	-14.318	8.336
Evoked Power (mV/Hz^2)			
Measure	**Mean (SD)**	**Minimum**	**Maximum**
Evoked Power (mV/Hz^2) at Baseline	0.385 (0.497)	0.006	3.613
Evoked Power (mV/Hz^2) at 18-Month Follow-Up	0.297 (0.322)	0.006	1.684
Evoked Power (mV/Hz^2) at 36-Month Follow-Up	0.210 (0.221)	0.002	1.030
Total Power (mV/Hz^2)			
Measure	**Mean (SD)**	**Minimum**	**Maximum**
Total Power (mV/Hz^2) at Baseline	1.738 (1.001)	0.344	5.941
Total Power (mV/Hz^2) at 18-Month Follow-Up	1.658 (0.759)	0.640	5.844
Total Power (mV/Hz^2) at 36-Month Follow-Up	1.574 (0.650)	0.859	3.570
Inter-Trial Phase Synchrony (ITPS)			
Measure	**Mean (SD)**	**Minimum**	**Maximum**
ITPS at Baseline	5.557 (0.442)	4.828	7.674
ITPS at 18-Month Follow-Up	5.446 (0.466)	4.255	7.121
ITPS at 36-Month Follow-Up	5.324 (0.343)	4.908	6.300

Table 1 (cont'd)

Inter-Channel Phase Synchrony (ICPS)			
Measure	**Mean (SD)**	**Minimum**	**Maximum**
ICPS between FCz and F3 at Baseline	4.673 (0.180)	4.141	5.450
ICPS between FCz and F3 at 18-Month Follow-Up	4.681 (0.177)	4.173	5.416
ICPS between FCz and F3 at 36-Month Follow-Up	4.665 (0.149)	4.219	4.916
ICPS between FCz and F5 at Baseline	4.702 (0.182)	4.356	5.287
ICPS between FCz and F5 at 18-Month Follow-Up	4.668 (0.153)	4.333	5.186
ICPS between FCz and F5 at 36-Month Follow-Up	4.743 (0.152)	4.569	5.103
ICPS between FCz and F4 at Baseline	4.694 (0.222)	3.960	5.781
ICPS between FCz and F4 at 18-Month Follow-Up	4.691 (0.165)	4.125	5.367
ICPS between FCz and F4 at 36-Month Follow-Up	4.706 (0.147)	4.409	5.063
ICPS between FCz and F6 at Baseline	4.728 (0.248)	4.268	6.507
ICPS between FCz and F6 at 18-Month Follow-Up	4.705 (0.204)	4.220	5.447

Table 1 (cont'd)

ICPS between FCz and F6 at 36-Month Follow-Up	4.707 (0.138)	4.423	5.101
Notes: RCADS = Revised Child Anxiety and Depression Scale; GAD = Generalized Anxiety Disorder; ITPS= Intertrial Phase Synchrony; ICPS = Interchannel Phase Synchrony			

Table 2: *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Error-Related Negativity (ERN) and Its Moderation by Baseline Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	-4.223	0.615	163.000	-6.870	<0.001
Baseline Anxiety	2.202	1.228	163.000	1.794	0.075
Baseline Age	-0.668	0.254	163.000	-2.633	0.009
Baseline Anxiety x Baseline Age	0.139	0.498	163.000	0.279	0.781
Recruitment Sample	0.763	0.625	163.000	1.220	0.224
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Family ID	0.00		0.00		
Residual	59.2		0.77		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	-4.358	0.642	163.000	-6.784	<0.001
Baseline Anxiety	0.976	1.092	163.000	0.893	0.373
Baseline Age	-0.681	0.269	163.000	-2.536	0.012
Baseline Anxiety x Baseline Age	0.383	0.487	163.000	0.786	0.433
Recruitment Sample	0.792	0.631	163.000	1.254	0.212
Random Effects					

Table 2 (cont'd)

Effect	***Variance***	***SD***
Intercept for Family ID	0.00	0.00
Residual	59.87	7.74
Notes: The amount of variance explained by the intercept for family was too small to be estimated. GAD Model: $R^2 = 0.027$; Social Anxiety Model: $R^2 = 0.016$		

Table 3: *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Error-Related Negativity (ERN) and Its Moderation by Change in Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	-4.076	0.575	276.059	-7.085	<0.001
Change in Anxiety	2.671	4.213	251.863	0.6634	0.527
Change in Age	0.472	0.374	190.482	1.264	0.208
Change in Anxiety x Change in Age	-2.091	2.078	240.722	-1.006	0.315
Baseline Age	-0.596	0.195	174.957	-3.062	0.003
Recruitment Sample	0.318	0.499	174.483	0.637	0.524
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Child ID	14.87		3.856		
Intercept for Family ID	0.00[x]		0.000		
Residual	40.32		6.350		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	-4.082	0.576	275.777	-7.084	<0.001
Change in Anxiety	-0.986	5.051	246.476	-0.195	0.845
Change in Age	0.483	0.384	190.837	1.257	0.210

Table 3 (cont'd)

Change in Anxiety x Change in Age	-0.199	22.291	235.147	-0.087	0.931
Baseline Age	-0.618	0.197	176.848	-3.133	0.002
Recruitment Sample	0.339	0.500	174.596	0.677	0.499

Random Effects		
Effect	***Variance***	***SD***
Intercept for Child ID	14.98	3.871
Intercept for Family ID	0.00[x]	0.000
Residual	40.35	6.352

Notes: [x] The amount of variance explained by the intercept for family was too small to be estimated. GAD Model: $ICC_{ChildID}$ = 0.269, R^2 = 0.020 ; Social Anxiety Model: $ICC_{ChildID}$ = 0.270, R^2 = 0.019

Table 4: *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Evoked Theta Power and Its Moderation by Baseline Age*

Model for GAD Symptoms

Fixed Effects

Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	0.384	0.040	88.394	9.531	<0.001
Baseline Anxiety	-0.029	0.079	147.521	-0.366	0.715
Baseline Age	0.046	0.016	161.509	2.857	0.005
Baseline Anxiety x Baseline Age	-0.033	0.031	159.079	-1.048	0.296
Recruitment Sample	-0.007	0.041	112.672	-0.171	0.865

Random Effects

Effect	***Variance***	***SD***
Intercept for Family ID	0.021	0.145
Residual	0.217	0.465

Model for Social Anxiety Symptoms

Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	0.398	0.041	122.021	9.659	<0.001
Baseline Anxiety	-0.028	0.069	160.171	-0.405	0.686
Baseline Age	0.045	0.017	161.914	2.695	0.008
Baseline Anxiety x Baseline Age	-0.042	0.030	160.265	-1.378	0.170
Recruitment Sample	-0.011	0.041	113.918	-0.286	0.779

Random Effects

Table 4 (cont'd)

Effect	***Variance***	***SD***
Intercept for Family ID	0.018	0.133
Residual	0.218	0.467
Notes: Two participants were removed from these analyses because their data was influential; GAD = Generalized Anxiety Disorder; GAD Model**:** ICC = 0.059, R^2 = 0.046; Social Anxiety Model: ICC = 0.075, R^2 = 0.040		

Table 5: *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Evoked Theta Power and Its Moderation by Change in Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	0.360	0.033	178.958	10.956	<0.001
Change in Anxiety	-0.090	0.230	233.197	-0.392	0.696
Change in Age	-0.033	0.020	175.209	-1.619	0.107
Change in Anxiety x Change in Age	0.072	0.113	220.945	0.638	0.524
Baseline Age	0.038	0.011	169.429	3.484	0.001
Recruitment Sample	0.002	0.029	115.363	0.078	0.938
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Child ID	0.044		0.210		
Intercept for Family ID	0.012		0.110		
Residual	0.115		0.339		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	0.360	0.033	177.439	10.939	<0.001
Change in Anxiety	0.153	0.275	231.734	0.557	0.578
Change in Age	-0.031	0.021	176.183	-1.506	0.134

Table 5 (cont'd)

Change in Anxiety x Change in Age	-0.049	0.124	222.979	-0.390	0.697
Baseline Age	0.039	0.011	176.003	3.528	0.001
Recruitment Sample	0.002	0.029	118.136	0.072	0.943

Random Effects		
Effect	***Variance***	***SD***
Intercept for Child ID	0.045	0.213
Intercept for Family ID	0.012	0.109
Residual	0.115	0.339

Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** $ICC_{ChildID}$ = 0.277, $ICC_{FamilyID}$ = 0.094, R^2 = 0.016; Social Anxiety Model: $ICC_{ChildID}$ = 0.281, $ICC_{FamilyID}$ = 0.094, R^2 = 0.021

Table 6: *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Theta Total Power and Its Moderation by Baseline Age*

Model for GAD Symptoms

Fixed Effects

Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	1.697	0.085	118.843	20.074	<0.001
Baseline Anxiety	-0.044	0.163	154.740	-0.267	0.790
Baseline Age	0.001	0.032	159.996	0.029	0.977
Baseline Anxiety x Baseline Age	-0.003	0.063	156.747	-0.048	0.962
Recruitment Sample	-0.127	0.086	121.070	-1.485	0.140

Random Effects

Effect	***Variance***	***SD***
Intercept for Family ID	0.181	0.425
Residual	0.821	0.906

Model for Social Anxiety Symptoms

Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	1.709	0.087	127.647	19.670	<0.001
Baseline Anxiety	-0.080	0.142	161.764	-0.565	0.573
Baseline Age	0.003	0.034	160.700	0.090	0.929
Baseline Anxiety x Baseline Age	-0.035	0.062	158.503	-0.566	0.572
Recruitment Sample	-0.132	0.086	121.866	-1.531	0.128

Random Effects

Table 6 (cont'd)

Effect	***Variance***	***SD***
Intercept for Family ID	0.181	0.425
Residual	0.817	0.904
Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** ICC = 0.181, R^2 = 0.001; Social Anxiety Model: ICC = 0.181, R^2 = 0.006		

Table 7: *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Total Theta Power and Its Moderation by Change in Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	1.669	0.074	163.957	22.712	<0.001
Change in Anxiety	-0.197	0.431	179.729	-0.457	0.649
Change in Age	0.008	0.037	148.756	-0.457	0.823
Change in Anxiety x Change in Age	0.056	0.210	168.445	0.268	0.789
Baseline Age	-0.004	0.025	176.659	-0.159	0.874
Recruitment Sample	-0.130	0.069	126.332	-1.883	0.062
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Child ID	0.317		0.563		
Intercept for Family ID	0.145		0.381		
Residual	0.348		0.590		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***Df***	***T value***	***p-value***
Intercept	1.669	0.073	163.712	22.764	<0.001
Change in Anxiety	-0.405	0.518	189.047	-0.781	0.436
Change in Age	0.001	0.004	148.800	0.016	0.987

Table 7 (cont'd)

Change in Anxiety x Change in Age	0.189	0.232	177.510	0.812	0.418
Baseline Age	-0.004	0.025	178.783	-0.169	0.866
Recruitment Sample	-0.132	0.234	125.821	-1.922	0.057

Random Effects		
Effect	***Variance***	***SD***
Intercept for Child ID	0.312	0.559
Intercept for Family ID	0.144	0.379
Residual	0.351	0.592

Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** $ICC_{ChildID}$ = 0.477, $ICC_{FamilyID}$ = 0.292, R^2 = 0.004; Social Anxiety Model: $ICC_{ChildID}$ = 0.471, $ICC_{FamilyID}$ = 0.291, R^2 = 0.000

Table 8: *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Theta Intertrial Phase Synchrony (ITPS) and Its Moderation by Baseline Age*

Model for GAD Symptoms

Fixed Effects

Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	5.558	0.035	93.927	160.865	<0.001
Baseline Anxiety	0.020	0.068	137.535	0.295	0.768
Baseline Age	0.059	0.014	161.545	4.192	<0.001
Baseline Anxiety x Baseline Age	-0.003	0.027	158.542	-0.112	0.911
Recruitment Sample	0.002	0.035	96.067	0.057	0.955

Random Effects

Effect	***Variance***	***SD***
Intercept for Family ID	0.010	0.100
Residual	0.170	0.412

Model for Social Anxiety Symptoms

Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	5.564	0.036	107.168	158.128	<0.001
Baseline Anxiety	-0.025	0.060	158.178	-0.419	0.676
Baseline Age	0.059	0.015	161.744	4.025	<0.001
Baseline Anxiety x Baseline Age	-0.017	0.027	159.505	-0.668	0.505
Recruitment Sample	0.000	0.035	97.224	-0.015	0.988

Random Effects

Table 8 (cont'd)

Effect	***Variance***	***SD***
Intercept for Family ID	0.009	0.095
Residual	0.170	0.413
Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** ICC = 0.056, R^2 = 0.102; Social Anxiety Model: ICC = 0.051, R^2 = 0.116		

Table 9: *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Theta Intertrial Phase Synchrony (ITPS) and Its Moderation by Change in Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	5.529	0.032	186.239	171	<0.001
Change in Anxiety	0.363	0.240	275.303	1.511	0.132
Change in Age	-0.040	0.022	210.592	-1.857	0.065
Change in Anxiety x Change in Age	-0.116	0.119	273.476	-0.977	0.330
Baseline Age	0.067	0.010	170.318	6.469	<0.001
Recruitment Sample	0.030	0.027	114.950	1.102	0.273
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Child ID	0.019		0.137		
Intercept for Family ID	0.008		0.092		
Residual	0.142		0.377		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	5.529	0.032	188.245	170	<0.001
Change in Anxiety	0.049	0.288	271.949	0.171	0.171
Change in Age	-0.046	0.022	214.723	-2.030	0.044

Table 9 (cont'd)

Change in Anxiety x Change in Age	0.059	0.131	265.825	0.450	0.653
Baseline Age	0.069	0.010	169.960	6.665	6.665
Recruitment Sample	0.028	0.027	117.066	1.042	0.300

Random Effects		
Effect	***Variance***	***SD***
Intercept for Child ID	0.015	0.123
Intercept for Family ID	0.009	0.096
Residual	0.144	0.380

Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** $ICC_{ChildID}$ = 0.118, $ICC_{FamilyID}$ =0.053, R^2 = 0.044; Social Anxiety Model: $ICC_{ChildID}$ = 0.094, $ICC_{FamilyID}$ = 0.059, R^2 = 0.031

Table 10. *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Theta Interchannel Phase Synchrony (ICPS) between FCz and Left Frontal Sites (F3 and F5) and Its Moderation by Baseline Age*

Model for GAD Symptoms

	FCz – F3					**FCz – F5**				
	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***Df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.676	0.002	53.164	323	<0.001	4.704	0.015	84.219	323	<0.001
Baseline Anxiety	0.009	0.003	105.913	0.328	0.743	0.028	0.029	127.256	0.949	0.344
Baseline Age	0.009	0.006	159.549	1.594	0.113	0.007	0.006	162.296	1.207	0.229
Baseline Anxiety x Baseline Age	-0.024	0.012	152.095	-2.108	0.037	-0.015	0.012	159.676	-1.244	0.216
Recruit Sample	0.003	0.015	55.061	0.172	0.864	0.001	0.015	86.081	0.073	0.942
	Random Effects					**Random Effects**				
Effect	***Variance***		***SD***			***Variance***		***SD***		
Intercept for Family ID	0.000		0.004			0.000		0.016		
Residual	0.030		0.174			0.033		0.181		

Model for Social Anxiety Symptoms

	FCz – F3	**FCz – F5**

Table 10 (cont'd)

	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***Df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.682	0.015	163.000	317	<0.001	4.706	0.015	95.991	309	<0.001
Baseline Anxiety	-0.014	0.025	163.000	-0.553	0.581	0.032	0.026	155.590	1.245	0.215
Baseline Age	0.010	0.006	163.000	1.548	0.123	0.005	0.006	161.835	0.850	0.397
Baseline Anxiety x Baseline Age	-0.021	0.011	163.000	-1.887	0.061	-0.009	0.011	159.216	-0.814	0.417
Recruitment Sample	0.001	0.015	163.000	0.006	0.947	0.000	0.015	84.120	0.008	0.993

	Random Effects		**Random Effects**	
Effect	***Variance***	***SD***	***Variance***	***SD***
Intercept for Family ID	0.000*	0.000	0.001	0.028
Residual	0.032	0.178	0.032	0.180

Notes: GAD = Generalized Anxiety Disorder; *FCz – F3:* GAD Model**:** ICC = 0.046; R^2 = 0.044; Social Anxiety Model: R^2 = 0.013*; *FCz – F5:* GAD Model**:** ICC = 0.008, R^2 = 0.003; Social Anxiety Model: ICC = 0.024, R^2 = 0.017
*The amount of variance explained by the intercept for family was too small to be estimated for this model.

Table 11. *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Theta Interchannel Phase Synchrony (ICPS) between FCz and Right Frontal Sites (F4 and F6) and Its Moderation by Baseline Age*

Model for GAD Symptoms

	FCz – F4					**FCz – F6**				
	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.699	0.017	163.000	269	<0.001	4.730	0.020	100.586	236	<0.001
Baseline Anxiety	-0.022	0.035	163.000	-0.624	0.534	-0.030	0.040	141.727	-0.755	0.452
Baseline Age	0.022	0.007	163.000	3.073	0.003	0.021	0.008	161.560	2.628	0.009
Baseline Anxiety x Baseline Age	0.005	0.014	163.000	0.349	0.728	-0.000	0.016	158.825	-0.019	0.985
Recruit Sample	0.023	0.018	163.000	1.318	0.189	0.012	0.020	102.714	0.595	0.553
	Random Effects					**Random Effects**				
Effect	***Variance***		***SD***			***Variance***		***SD***		
Intercept for Family ID	0.000*		0.000			0.004		0.063		
Residual	0.048		0.218			0.056		0.237		
Model for Social Anxiety Symptoms										
	FCz – F4					**FCz – F6**				

Table 11 (cont'd)

	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***Df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.698	0.018	163.000	260	<0.001	4.731	0.021	113.455	228	<0.001
Baseline Anxiety	-0.039	0.031	163.000	-1.267	0.207	-0.040	0.035	159.107	-1.142	0.255
Baseline Age	0.245	0.008	163.000	3.270	0.001	0.023	0.009	161.786	2.765	0.006
Baseline Anxiety x Baseline Age	0.004	0.014	163.000	0.322	0.748	-0.001	0.015	159.787	-0.093	0.926
Recruitment Sample	0.024	0.018	163.000	1.343	0.181	0.012	0.020	104.229	0.600	0.550
	Random Effects					**Random Effects**				
Effect	***Variance***		***SD***			***Variance***		***SD***		
Intercept for Family ID	0.000*		0.000*			0.004		0.063		
Residual	0.047		0.218			0.056		0.237		

Notes: GAD = Generalized Anxiety Disorder; *FCz – F4:* GAD Model**:** R^2 = 0.030; Social Anxiety Model: R^2 = 0.037; *FCz – F6:* GAD Model**:** ICC = 0.066, R^2 = 0.050; Social Anxiety Model: ICC = 0.062, R^2 = 0.050
*The amount of variance explained by the intercept for family was too small to be estimated for this model.

Table 12. *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Theta Interchannel Phase Synchrony (ICPS) between FCz and Left Frontal Sites (F3 and F5) and Its Moderation by Change in Age*

Model for GAD Symptoms

	FCz – F3					**FCz – F5**				
	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***Df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.670	0.014	285.676	346	0.794	4.694	0.013	213.112	354	<0.001
Change in Anxiety	0.027	0.102	278.327	0.262	0.794	0.094	0.100	294.944	0.940	0.348
Change in Age	0.006	0.009	213.802	0.600	0.549	0.002	0.009	276.459	0.262	0.793
Change in Anxiety x Change in Age	-0.024	0.051	273.909	-0.466	0.642	-0.035	0.049	291.602	-0.714	0.476
Baseline Age	0.012	0.004	173.570	2.609	0.010	0.011	0.004	245.888	2.783	0.006
Recruitment Sample	0.002	0.011	174.868	0.193	0.847	0.023	0.011	107.554	2.162	0.033
	Random Effects					**Random Effects**				
Effect	***Variance***		***SD***			***Variance***		***SD***		
Intercept for Child ID	0.005		0.069			0.000 [x]		0.000		

Table 12 (cont'd)

Intercept for Family ID	0.000 [x]	0.000	0.002	0.038
Residual	0.026	0.161	0.027	0.165

Model for Social Anxiety Symptoms

	FCz – F3					**FCz – F5**				
	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***Df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.670	0.014	285.987	345	<0.001	4.694	0.013	216.170	355	<0.001
Change in Anxiety	-0.027	0.123	270.139	-0.221	0.826	-0.031	0.121	290.556	-0.255	0.0799
Change in Age	0.005	0.010	216.094	0.544	0.587	0.002	0.010	278.749	0.220	0.826
Change in Anxiety x Change in Age	0.007	0.056	262.941	0.128	0.898	0.755	0.055	287.916	0.313	0.755
Baseline Age	0.011	0.005	175.430	2.540	0.012	0.011	0.011	246.886	2.712	0.007
Recruitment Sample	0.002	0.011	175.580	0.202	0.840	0.023	0.023	110.723	2.175	0.032
	Random Effects					**Random Effects**				
Effect	***Variance***		***SD***			***Variance***		***SD***		

Table 12 (cont'd)

Intercept for Child ID	0.005	0.068	0.000 [x]	0.000
Intercept for Family ID	0.000 [x]	0.000	0.001	0.034
Residual	0.026	0.161	0.027	0.165

Notes: GAD = Generalized Anxiety Disorder; *FCz – F3:* GAD Model**:** $ICC_{ChildID}$ = 0.153; R^2 = 0.000; Social Anxiety Model: $ICC_{FamilyID}$ = 0.161, R^2 = 0.00; *FCz – F5:* GAD Model**:** $ICC_{ChildID}$ = 0.049; R^2 = 0.020; Social Anxiety Model: $ICC_{FamilyID}$ = 0.044, R^2 = 0.010

[x] The amount of variance explained by the intercept was too small to be estimated.

Table 13. *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Theta Interchannel Phase Synchrony (ICPS) between FCz and Right Frontal Sites (F4 and F6) and Its Moderation by Change in Age*

Model for GAD Symptoms										
	FCz – F4					**FCz – F6**				
	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***Df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.688	0.015	295.000	315	<0.001	4.722	0.017	180.953	272.97	<0.001
Change in Anxiety	-0.144	0.115	295.000	-1.244	0.214	0.048	0.132	285.545	0.367	0.714
Change in Age	0.008	0.011	295.000	0.732	0.465	-0.004	0.012	229.584	-0.310	0.757
Change in Anxiety x Change in Age	0.073	0.057	295.000	1.278	0.202	-0.017	0.065	290.402	-0.265	0.791
Baseline Age	0.016	0.005	295.000	3.466	0.001	0.015	0.006	172.205	2.707	0.008
Recruitment Sample	0.013	0.012	295.000	1.077	0.282	0.011	0.014	104.583	0.777	0.439
	Random Effects					**Random Effects**				
Effect	***Variance***		***SD***			***Variance***		***SD***		
Intercept for Child ID	0.000 [x]		0.000			0.003		0.056		

Table 13 (cont'd)

Intercept for Family ID	0.000 [x]	0.000	0.001	0.025
Residual	0.038	0.194	0.046	0.215

Model for Social Anxiety Symptoms

	FCz – F4					**FCz – F6**				
	Fixed Effects					**Fixed Effects**				
Effect	***B***	***SE***	***Df***	***T value***	***p-value***	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	4.688	0.015	295.000	313	<0.001	4.722	0.017	180.949	273	<0.001
Change in Anxiety	0.042	0.139	295.000	0.300	0.764	0.090	0.159	283.411	0.568	0.571
Change in Age	0.007	0.011	295.000	0.612	0.541	-0.001	0.012	232.610	-0.095	0.925
Change in Anxiety x Change in Age	-0.000	0.064	295.000	-0.007	0.995	-0.050	0.072	280.393	-0.691	0.490
Baseline Age	0.017	0.005	295.000	3.600	<0.001	0.015	0.006	173.895	2.655	0.009
Recruitment Sample	0.012	0.012	295.000	1.022	0.308	0.012	0.014	106.077	0.829	0.409

	Random Effects		**Random Effects**	
Effect	***Variance***	***SD***	***Variance***	***SD***

Table 13 (cont'd)

Intercept for Child ID	0.000 [x]	0.000	0.003	0.051
Intercept for Family ID	0.000 [x]	0.000	0.001	0.024
Residual	0.038	0.194	0.046	0.216

Notes: GAD = Generalized Anxiety Disorder; *FCz – F3:* GAD Model**:** *ICCs* = 0.000, R^2 = 0.047; Social Anxiety Model: *ICCs* = 0.000, R^2 = 0.044; *FCz – F5:* GAD Model**:** $ICC_{ChildID}$ = 0.063, $ICC_{FamilyID}$ = 0.007, R^2 = 0.006 ;Social Anxiety Model: $ICC_{ChildID}$ = 0.060, $ICC_{FamilyID}$ = 0.006, R^2 = 0.005

[x] The amount of variance explained by the intercept was too small to be estimated.

Table 14: *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Number of No-Go Errors Made and Its Moderation by Baseline Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	25.234	0.704	101.425	35.824	<0.001
Baseline Anxiety	-0.331	1.386	143.513	-0.239	0.812
Baseline Age	-1.567	0.281	161.153	-5.573	<0.001
Baseline Anxiety x Baseline Age	-0.392	0.551	158.113	-0.713	0.477
Recruitment Sample	1.722	0.716	103.652	2.407	0.018
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Family ID	6.282		2.506		
Residual	67.157		8.195		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	25.101	0.731	113.766	34.331	<0.001
Baseline Anxiety	0.477	1.216	159.765	0.392	0.696
Baseline Age	-1.556	0.296	161.230	-5.258	<0.001
Baseline Anxiety x Baseline Age	0.315	0.535	158.881	0.589	0.557

Table 14 (cont'd)

Recruitment Sample	1.778	0.722	105.228	2.463	0.015
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Family ID	7.128		2.670		
Residual	66.397		8.148		
Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** ICC = 0.086, R^2 = 0.133; Social Anxiety Model: ICC = 0.097, R^2 = 0.145					

Table 15: *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Number of No-Go Errors Made and Its Moderation by Change in Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	25.621	0.687	163.764	37.316	<0.001
Change in Anxiety	-3.189	4.537	210.874	-0.703	0.483
Change in Age	-2.539	0.395	161.510	-6.433	<0.001
Change in Anxiety x Change in Age	1.115	2.217	198.817	0.503	0.616
Baseline Age	-1.232	0.228	165.934	-5.404	<0.001
Recruitment Sample	1.733	0.622	114.884	2.785	0.006
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Child ID	20.554		4.534		
Intercept for Family ID	9.468		3.077		
Residual	42.259		6.501		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	25.631	0.685	164.934	37.420	<0.001
Change in Anxiety	-2.467	5.429	217.127	-0.454	0.650
Change in Age	-2.488	0.408	162.684	-6.100	<0.001

Table 15 (cont'd)

Change in Anxiety x Change in Age	0.119	2.452	204.392	0.049	0.961
Baseline Age	-1.266	0.228	166.018	-5.550	<0.001
Recruitment Sample	1.741	0.619	115.314	2.811	0.006

Random Effects		
Effect	***Variance***	***SD***
Intercept for Child ID	19.082	4.368
Intercept for Family ID	9.729	3.119
Residual	42.854	6.546

Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** $ICC_{ChildID}$ = 0.327, $ICC_{FamilyID}$ = 0.183, R^2 = 0.250; Social Anxiety Model: $ICC_{ChildID}$ = 0.308, $ICC_{FamilyID}$ = 0.187 , R^2 = 0.239

Table 16: *Estimates from Multilevel Models Examining the Association between Baseline Anxiety and Go Correct Average Reaction Time and Its Moderation by Baseline Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	489.931	4.142	99.159	118.274	<0.001
Baseline Anxiety	-1.246	8.038	147.610	-0.155	0.877
Baseline Age	-20.611	1.606	158.786	-12.833	<0.001
Baseline Anxiety x Baseline Age	-1.280	3.139	153.969	-0.408	0.684
Recruitment Sample	1.497	4.205	101.772	0.356	0.723
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Family ID	383		19.57		
Residual	2052		45.30		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	488.199	4.246	111.109	114.972	<0.001
Baseline Anxiety	-8.876	6.922	160.942	-1.282	0.202
Baseline Age	-19.454	1.668	159.132	-11.663	<0.001
Baseline Anxiety x Baseline Age	5.285	3.011	155.517	1.755	0.081
Recruitment Sample	2.221	4.203	103.894	0.529	0.598
Random Effects					

Table 16 (cont'd)

Effect	***Variance***	***SD***
Intercept for Family ID	433.3	20.81
Residual	1948.8	44.15
Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** ICC = 0.157, R^2 = 0.495; Social Anxiety Model: ICC = 0.182, R^2 = 0.521		

Table 17: *Estimates from Multilevel Models Examining the Association between Change in Anxiety and Go Correct Average Reaction Time and Its Moderation by Change in Age*

Model for GAD Symptoms					
Fixed Effects					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	498.380	3.827	141.347	130.241	<0.001
Change in Anxiety	-12.862	26.577	217.223	-0.484	0.629
Change in Age	-23.918	2.326	160.755	-10.281	<0.001
Change in Anxiety x Change in Age	5.534	13.041	203.897	0.424	0.672
Baseline Age	-19.727	1.295	160.356	-15.235	<0.001
Recruitment Sample	0.282	3.409	92.353	0.083	0.934
Random Effects					
Effect	***Variance***		***SD***		
Intercept for Child ID	710.5		22.66		
Intercept for Family ID	133.9		11.57		
Residual	1503.2		38.77		
Model for Social Anxiety Symptoms					
Effect	***B***	***SE***	***df***	***T value***	***p-value***
Intercept	498.051	3.755	146.001	133	<0.001
Change in Anxiety	17.352	31.778	224.249	0.546	0.586
Change in Age	-24.352	2.398	166.780	-10.204	<0.001

Table 17 (cont'd)

Change in Anxiety x Change in Age	0.662	14.383	210.749	0.046	0.963
Baseline Age	-19.405	1.291	165.349	-15.034	<0.001
Recruitment Sample	0.067	3.325	93.416	0.202	0.984

Random Effects		
Effect	***Variance***	***SD***
Intercept for Child ID	709.29	26.633
Intercept for Family ID	66.73	8.169
Residual	1525.97	39.064

Notes: GAD = Generalized Anxiety Disorder; GAD Model**:** : $ICC_{ChildID}$ = 0.321, $ICC_{FamilyID}$ = 0.082, R^2 = 0.451; Social Anxiety Model: $ICC_{ChildID}$ = 0.317, $ICC_{FamilyID}$ = 0.042, R^2 = 0.441

Supplementary Exploratory Correlations

Correlations between dependent variables are presented in Table A1. Unexpectedly, number of errors made was not significantly associated with the ERN amplitude or ICPS. However, number of errors was significantly negatively associated with power and ITPS as expected. Reaction time was significantly associated with the ERN, evoked power, ITPS and ICPS between FCz and right frontolateral sites. Unexpectedly, reaction time was not significantly associated with total power or ICPS between FCz and left frontolateral sites. As expected, a larger ERN was associated with greater power and ITPS. Contrary to expectations, the ERN amplitude was only associated with greater ICPS between FCz and F5. In line with expectations, greater evoked power was associated with greater total power, greater ITPS and greater functional connectivity for the majority of ICPS measures (although not between FCz and F5). Notably, greater total power was only significantly associated with greater ITPS and greater ICPS between FCz and right frontolateral sites, contrary to expectations. Greater ITPS was associated with greater functional connectivity for the majority of ICPS measures (although not between FCz and F5). ICPS measures were all significantly related, although the strength of these associations varied based on lateralization.

Additionally, GAD and Social Anxiety subscale scores at baseline were positively and strongly correlated ($r = 0.648$ $p < 0.001$). Baseline age was positively correlated with social anxiety ($r = 0.299$, $p < 0.001$) and was not significantly correlated with GAD ($r = 0.063$, $p = 0.416$).

Table A1: *Exploratory Correlations Between Dependent Variables at Baseline*

Variables	2	3	4	5	6	7	8	9	10
1.Number of No-Go Errors	0.12	0.14	**-0.30***	**-0.16***	**-0.24***	-0.10	-0.05	-0.07	-0.12
2. Go Correct Reaction Time at Baseline (ms)	-	**0.25***	**-0.21***	-0.08	**-0.31***	-0.14	**-0.20***	-0.11	**-0.19***
3. ERN Amplitude (mV) at Baseline	-	-	**-0.53***	**-0.48***	**-0.35***	-0.09	**-0.20***	-0.06	-0.12
4. Evoked Power (mV/Hz^2) at Baseline	-	-	-	**0.74***	**0.59***	**0.23***	**0.31***	0.11	**0.27***
5. Total Power (mV/Hz^2) at Baseline	-	-	-	-	**0.50***	0.13	**0.23***	0.08	**0.20***
6. ITPS at Baseline	-	-	-	-	-	**0.18***	**0.29***	0.12	**0.23***
7. ICPS between FCz and F3 at Baseline	-	-	-	-	-	-	**0.22***	**0.48***	**0.29***
8. ICPS between FCz and F4 at Baseline	-	-	-	-	-	-	-	**0.23***	**0.67***
9. ICPS between FCz and F5 at Baseline	-	-	-	-	-	-	-	-	**0.24***
10. ICPS between FCz and F6 at Baseline	-	-	-	-	-	-	-	-	-

Notes: * indicates $p < 0.05$; ITPS= Intertrial Phase Synchrony; ICPS = Interchannel Phase Synchrony